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# ENVIRONMENTAL ASSESSMENT BOARD

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VOLUME: 214

DATE: Wednesday, June 13, 1990

BEFORE:

A. KOVEN, Chairman

E. MARTEL, Member



FOR HEARING UPDATES CALL (TOLL-FREE): 1-800-387-8810

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# ENVIRONMENTAL ASSESSMENT BOARD

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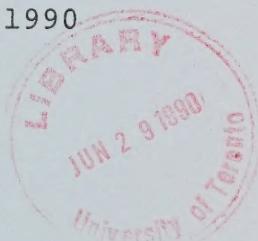
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HEARING ON THE PROPOSAL BY THE MINISTRY OF NATURAL  
RESOURCES FOR A CLASS ENVIRONMENTAL ASSESSMENT FOR  
TIMBER MANAGEMENT ON CROWN LANDS IN ONTARIO

IN THE MATTER of the Environmental  
Assessment Act, R.S.O. 1980, c.140;

- and -

IN THE MATTER of the Class Environmental  
Assessment for Timber Management on Crown  
Lands in Ontario;

- and -

IN THE MATTER OF a Notice by the  
Honourable Jim Bradley, Minister of the  
Environment, requiring the Environmental  
Assessment Board to hold a hearing with  
respect to a Class Environmental  
Assessment (No. NR-AA-30) of an  
undertaking by the Ministry of Natural  
Resources for the activity of timber  
management on Crown Lands in Ontario.

Hearing held at the offices of the Ontario  
Highway Transport Commission, Britannica  
Building, 151 Bloor Street West, 10th Floor,  
Toronto, Ontario, on Wednesday, June  
13th, 1990, commencing at 8:30 a.m.

VOLUME 214

BEFORE:

MRS. ANNE KOVEN  
MR. ELIE MARTEL

Chairman  
Member



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I N D E X   o f   P R O C E E D I N G S

<u>Witnesses:</u>	<u>Page No.</u>
<u>JOSEPH V. RODRICKS,</u> <u>NANCY J RACHMAN, Sworn</u>	38477
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Cross-examination by Mr. Castrilli	38605



I N D E X   o f   E X H I B I T S

<u>No.</u>	<u>Description</u>	<u>Page No.</u>
1239	Witness statement of Panel 9B.	38475
1240	Hard copy of overheads to be referred to by Dr. Rachman.	38479
1241	Hard copy of overheads to be referred to by Dr. Rodricks.	38479
1242	Document entitled Notice: Status of Consideration for a Special Review.	38531
1243	Document entitled EPA Updates List of Classified Carcinogenic Pesticides.	38539
1244	Document entitled Mortality Study of Canadian Male Farm Operators: Non-Hodgkin's Lymphoma Mortality and Agricultural Practices in Saskatchewan.	38542
1245	Document entitled The Weight of the Evidence of the Human Carcinogenicity of 2,4-D.	38568
1246	Document entitled Guidelines for Carcinogen Risk Assessment, dated September 24, 1986 published by the U.S. EPA.	38603
1247	Document entitled Soft Tissue Sarcoma and Non-Hodgkin's Lymphoma in Relation to Phenoxyherbicide and Chlorinated Phenol Exposure in Western Washington, by James S. Woods et al.	38650



I N D E X   o f   E X H I B I T S  
(Cont'd)

No.	Description	Page No.
1248	Editorial entitled Herbicides and Non-Hodgkin's Lymphoma, New Evidence From a study of Saskatchewan Farmers by Aaron Blair.	38703



1           ---Upon commencing at 8:30 a.m.

2           MADAM CHAIR: Good morning.

3           MR. CASSIDY: Good morning.

4           MADAM CHAIR: Please be seated.

5           Good morning, Mr. Cassidy.

6           MR. CASSIDY: Good morning. I am not  
7           Eleanor Cronk but I am prepared to proceed with the  
8           next panel, Panel 9B.

9           Madam Chair, the witnesses are here and I  
10          propose to commence by filing a copy of Panel 9B as the  
11          next exhibit. Perhaps you could tell me which number  
12          that would be, Madam Chair.

13          MADAM CHAIR: Exhibit 1239.

14          MR. CASSIDY: Thank you, Madam Chair.

15          ---EXHIBIT NO. 1239: Witness Statement of Panel 9B.

16          MR. CASSIDY: Madam Chair, the witnesses  
17          are present before you. They are Dr. Joe Rodricks and  
18          Dr. Nancy Rachman from Environ Corporation. I will ask  
19          shortly Dr. Rodricks to outline and give some  
20          background to Environ Corporation, but I first wish to  
21          advise the Board the reason for these witnesses  
22          appearing before you and specifically those reasons are  
23          outlined in paragraph 3 of the Executive Summary of  
24          Exhibit 1239, the witness statement, which can be found  
25          on (ii).

1                   Environ Corporation was requested to  
2 evaluate and review the relevant evidence before this  
3 Board and to advise the Industry and subsequently this  
4 Board regarding issues relating to (a) the process by  
5 which the environmental protection agency in the United  
6 States authorizes pesticides use in forestry as it  
7 concerns issues which have arisen in the course of  
8 evidence before this Board; (b) the evaluation of  
9 public health risks from the use in forestry of  
10 pesticides including sources of toxicity and data and  
11 risk assessment, and the evaluation of existing  
12 scientific evidence concerning the possible toxicity of  
13 the pesticides used in forestry and, in particular, Dr.  
14 Rodricks will be referring to two major exhibits before  
15 this Board dealing with that matter.

16                  That is all I propose to say by way of  
17 introduction, Madam Chair. I now propose to qualify  
18 Dr. Rachman and Dr. Rodricks as expert witnesses and I  
19 would ask that Dr. Rachman be qualified as an expert in  
20 the registration of pesticides in the United States and  
21 Dr. Rodricks be qualified as an expert witness in  
22 toxicology, specializing in human health risk  
23 assessment principles for chemicals.

24                  Both of the witnesses have asked to be  
25 affirmed before you this morning.

1                           JOSEPH V. RODRICKS,  
2                           NANCY J. RACHMAN, Affirmed

3                           MR. CASSIDY: As I indicated, Madam  
4                           Chair, I would like to ask Dr. Rodricks to provide a  
5                           brief description of Environ corporation. For your  
6                           benefit and the benefit of the parties, a complete  
7                           description of Environ Corporation can be found at  
8                           Appendix B of Exhibit 1239. The curriculum vitae for  
9                           both Dr. Rodricks and Dr. Rachman can be found as well  
10                          at Appendix B.

11                         The description of Environ Corporation  
12                         commences at page 28 of Appendix B of Exhibit 1239.

13                         DIRECT EXAMINATION BY MR. CASSIDY:

14                         Q. I would ask, Dr. Rodricks, if you  
15                         could give a brief overview of that description for the  
16                         benefit of the Board.

17                         DR. RODRICKS: A. Yes, thank you.  
18                         Environ Corporation is a technical consulting firm with  
19                         headquarters in Arlington, Virginia. We have offices  
20                         at three other locations in the United States as well.  
21                         We are just eight years in this business. We were  
22                         formed in 1982 to provide risk assessment services to  
23                         government, to industry.

24                         We gathered experts in all of the  
25                         disciplines related to risk assessment. My background

1       is in health sciences and I direct the health sciences  
2       operation of the company. We are mostly toxicologists,  
3       epidemiologists, scientists from the public health  
4       professions, about half of us, the 40 or so health  
5       scientists in the company are at the doctoral level or  
6       well beyond that.

7                   We also have an even larger staff of  
8       environmental scientists and environmental engineers,  
9       people who specialize in ground water, in air  
10      modelling, physical scientists and engineers, who deal  
11      with issues of human exposure to chemicals from many  
12      different sources.

13                  That staff puts together risk assessments  
14      and we have worked on almost every imaginable issue  
15      concerning chemicals in the environment or in the work  
16      place, occupational exposures as well. We have served  
17      also quite a wide variety of clients. We have worked  
18      for government in the U.S., for the environmental  
19      protection agency on occasion or the Occupational  
20      Safety and health Administration. We have also  
21      performed consulting of this type for a wide variety of  
22      industrial firms, both in the United States and abroad.

23                  I think that's a capsule of some of our  
24      work.

25                  Q. Thank you

1                   MR. CASSIDY: Now, Madam Chair, Mr.  
2 Martel, these witnessess will be referring to a number  
3 of overheads in the course of explaining their evidence  
4 this morning and I have prepared hard copies of these  
5 overheads which I now provide to the parties and file  
6 as the next exhibits.

7                   There are two sets of overheads; one set  
8 prepared for Dr. Rachman and another set prepared for  
9 Dr. Rodricks, and I would ask that they be filed as the  
10 next exhibits. I have collected the overheads in a  
11 stapled form and numbered -- each overhead numbered and  
12 in the course of their evidence, the witnesses will  
13 guide you through these overheads by referring to the  
14 page numbers on these exhibits.

15                  If we could then ask that the set of  
16 overheads prepared by Dr. Rachman be the next exhibit,  
17 which I believe would be exhibit 1240, and the  
18 overheads prepared by Dr. Rodricks be filed as Exhibit  
19 1241.

20                  I will now provide hard copies of those  
21 to the parties and the Board. (handed)

22                  MADAM CHAIR: Thank you.

23                  ---EXHIBIT NO. 1240: Hard copy of overheads to be  
24 referred to by Dr. Rachman  
25                  ---EXHIBIT NO. 1241: Hard copy of overheads to be  
                      referred to by Dr. Rodrick's

1                   MR. CASSIDY: I would like to commence  
2 this morning's evidence, Madam Chair, with Dr. Rachman  
3 who will be testifying, as is indicated in the  
4 Executive Summary, to Section 1 of the witness  
5 statement, Exhibit 1239. For the Board's benefit, for  
6 your notes, that section commences at page 4 of Exhibit  
7 1239 and concludes as at page 23 of the witness  
8 statement.

9                   As Dr. Rachman will be referring to a  
10 number of overheads, I will be going over periodically  
11 to turn them on.

12                  The first overhead, which could be found  
13 at page 1 of Exhibit 1240, will be where we will  
14 commence with Dr. Rachman.

15                  Q. I would ask you, Dr. Rachman, to  
16 please describe for the Board the legal criterion that  
17 is used for registration of pesticides in the United  
18 States.

19                  DR. RACHMAN: A. I'll be happy to do  
20 that. Would it be awkward for the Board if I stand?

21                  MADAM CHAIR: No.

22                  DR. RACHMAN: The registration of  
23 pesticides in the United States is under the authority  
24 of the Federal Insecticide, Fungicide and Rodenticide  
25 Act which is known as FIFRA. Section 3(c)(5) of FIFRA

1 provides the criteria which must be satisfied in order  
2 to register a pesticide.

3                   What I will do now is to paraphrase for  
4 you from the law, rather than quote. In order to  
5 register a pesticide, the administrator of the EPA must  
6 make a finding that the product's composition warrants  
7 the claims that are made for it; that the labeling  
8 that's submitted to support the registration, as well  
9 as other materials that are required by this section of  
10 the law are in compliance with the requirements of the  
11 law; that the product will perform its intended  
12 function without unreasonable adverse effects on the  
13 environment; and that when used according to widespread  
14 practice it will not generally cause unreasonable  
15 adverse effects on the environment.

16                   A couple of definitions here for your  
17 information. The term unreasonable adverse effects in  
18 the environment is defined to mean an unreasonable  
19 risk, taking into account not only risk information but  
20 also the economic, social and environmental costs or  
21 benefits of the pesticides use. So you will note that  
22 this is not a no-risk statute, that there is a risk  
23 benefit comparison that is specified by the law.

24                   The term environment in FIFRA includes  
25 air, water, land and all plants and animals living

1       therein, and also the inter-relationships between all  
2       of these living things. So that's a very broad  
3       definition of environment.

4                    MR. CASSIDY: Q. And those definitions  
5       are both contained on page 1 of Exhibit 1240, the  
6       overhead?

7                    DR. RACHMAN: A. Yes, that's correct.

8                    Now, in order to determine whether or not  
9       the products for registration meets these criteria, the  
10      EPA has the authority require data to be submitted.

11                  Q. And that is the subject of the next  
12      overhead?

13                  A. That's correct.

14                  MR. CASSIDY: Which is page 2 of Exhibit  
15      1240, Madam Chair.

16                  DR. RACHMAN: This overhead summarizes  
17      for you the general subject areas. Not the specific  
18      tests that are required, but the general subject areas  
19      in which data must be supplied and I will tell you in a  
20      general way what the EPA is looking for each in each  
21      one of these areas.

22                  Product chemistry. The registrant must  
23      determine the basic physical and chemical  
24      characteristics of the active ingredient and a  
25      formulated product and must determine what impurities

1       are present. Impurities that are present to a level of  
2       a tenth of a per cent or greater must be characterized.  
3       If the EPA decides that those impurities are of  
4       toxicological significance it has the authority to  
5       request the registrant to submit data on those  
6       impurities. The registrant must certify the upper and  
7       lower limits of active ingredient and impurities in the  
8       product as it will be sold.

9                          In the area of environmental fate, the  
10       idea is to establish the identity of degradates that  
11       form when the product is used, and also to characterize  
12       in a quantitative fashion the time course of the  
13       dissipation of the product in the environment.

14                          The toxicology area is the one that  
15       pertains to potential human health effects. These are  
16       tests of the inherent toxicity of the active ingredient  
17       or formulation and there are four general areas  
18       included. The short-term testing includes acute  
19       toxicity tests and tests of irritation and  
20       sensitization. Long-term testing, that category  
21       includes chronic feeding, oncogenicity, reproduction.  
22       Metabolism is generally a study done in a rat to  
23       determine how the pesticide is metabolized in a  
24       mammalian system and the identity and quantification of  
25       the metabolic products that are formed, and

1       genotoxicity is another word for mutagenicity studies.

2                     In the area of wildlife and aquatic  
3                     organisms, there the intent is to characterize the  
4                     inherent toxicity to wildlife, non-target organisms,  
5                     and some of these tests are also done on the formulated  
6                     product.

7                     The residue area involves determining the  
8                     nature and the magnitude of the residues that are  
9                     formed in the environment. This is particularly  
10                    important for pesticides that are applied to food crops  
11                    or applied to water.

12                  Now, there is a broad category called  
13                  special studies and in here I have lumped several other  
14                  requirements that may not be imposed on a routine  
15                  basis, but are required for certain products and  
16                  certain use pattern. Drift, for example, for certain  
17                  aerially applied products, specific measurements,  
18                  actual measurements of exposure under conditions of  
19                  product use may be required, depending on the  
20                  additional information that may come to the agency's  
21                  attention and I will talk a little bit more about that  
22                  in a minute.

23                  The specific tests that are required in  
24                  each one of these areas are determined in general by  
25                  the proposed use pattern for the product and

1 specifically the nature of the anticipated exposure  
2 from that proposed use pattern. Now, the specific  
3 tests are listed -- laid out in the U.S. Code of  
4 Federal Regulations, Volume 40. This is referred to as  
5 40 CFR.

6 MR. CASSIDY: Which is just at the bottom  
7 of that overhead.

8 DR. RACHMAN: Yes, I am sorry, you can't  
9 see it, but you will see it on the hand-out.

10 40 CFR, part 158. That part of the U.S.  
11 Code of Regulations contains charts and in each one of  
12 these subject areas it explains which tests are  
13 required depending upon what the proposed use pattern  
14 of the pesticide at issue will be.

15 Now, as I've said, the determination of  
16 what tests are required is based on the anticipated  
17 degree of exposure of the proposed use. In the United  
18 States, the food crop use is considered to have the  
19 widest potential exposure, the largest number of people  
20 are potentially exposed to residues of the pesticides  
21 if they are applied to growing food crops and,  
22 therefore, in that category, food use is subject to the  
23 most extensive data requirements.

24 I call that to your attention because of  
25 the chemical pesticides at issue here in this

1 proceeding, six of them are registered for food use in  
2 the United States and those are 2,4-D, glyphosate,  
3 hexazinone, picloram, simazine and carbaryl. I will  
4 return several times to that point and why that is of  
5 importance.

6 Now, some requirements are conditional.  
7 If you were to go to 40 CFR, part 158 and take a look  
8 at those charts that I mentioned, some of the  
9 requirements are indicated as conditional. That means  
10 that a decision is made by EPA as to whether that  
11 particular test is required on a case-by-case basis and  
12 those charts also lay out in a series of extensive  
13 footnotes the conditions under which those data may be  
14 required.

15 I might give you some examples for  
16 forestry use pesticides. Worker exposure requirements.  
17 The requirement for a specific test of worker exposure  
18 to a specific pesticide intended for forestry use will  
19 depend on EPA's judgment of the toxicity of the  
20 pesticide. They will look at the initial toxicity  
21 tests that are required and make the determination as  
22 to whether a test of that particular chemical under  
23 these conditions is needed.

24 The same thing is true of drift  
25 potential. If the pesticide is toxic or if for some

1 other reason the agency thinks that there may be some  
2 other potential to drift, maybe because of the physical  
3 chemical characteristics of the chemical or the  
4 formulation, EPA can require the registrant to do a  
5 drift study under actual use conditions.

6 That authority is fairly broad; that is,  
7 the authority to require additional information, and  
8 even tests that are not included in 40 CFR, Part 158  
9 have been required upon occasion; special testing that  
10 might be appropriate to particular chemicals.

11 Now, as I mentioned and as is indicated  
12 on this overhead, some tests are done on a formulated  
13 product, some tests are done on the active ingredient  
14 alone. As a general rule of thumb, I would say that  
15 the tests that are done to determine the inherent  
16 chemical and toxicological characteristics of the  
17 active ingredient are done on the active ingredient and  
18 in that area I would put the toxicology tests and some  
19 of the chemical tests. However, there are some  
20 wildlife toxicity tests that are also done on active  
21 ingredient and so on.

22 Generally, the tests that are trying to  
23 determine how the chemical will behave in the  
24 environment and the potential for human or non-target  
25 organism effects under actual conditions of use, those

1 tests are done on a formulated product as formulated  
2 for use, so that the results give as accurate a picture  
3 as possible of what the impacts of actual use of that  
4 product are going to be.

5 Now, as you know from previous testimony,  
6 the ingredients in a pesticide that are not active  
7 ingredients, that do not of themselves have pesticidal  
8 properties are called inert ingredients. What you  
9 should know is that in the United States inert  
10 ingredients, any ingredients in a product that is  
11 applied to growing food crops must either receive a  
12 tolerance, what is called in Canada a maximum residue  
13 limit, or must be specifically exempted from the  
14 requirement for such tolerance by the agency and this  
15 applies a review of safety information.

16 Chemicals that have met that criterion;  
17 that is, that have exempted from the requirement for  
18 tolerance are published in the Code of Federal  
19 Regulations at 40 CFR, Part 1(a).1001, one thousand and  
20 one. To the best of my knowledge, that list is the  
21 reference that manufacturers use when selecting  
22 ingredients to use in formulations for new products;  
23 not just not food use products, but all products and  
24 that is the best of my understanding.

25 Now, you are also aware from previous

1 evidence that in 1987 the EPA formulated a policy for  
2 regulating inert ingredient and I believe a copy of  
3 that policy had been submitted to you previously as  
4 Exhibit 725.

5 In developing this policy, the EPA  
6 promulgated four lists of inert ingredients, and I  
7 would like to explain to you a little bit, to the best  
8 of my knowledge, how those lists were generated. What  
9 EPA did was to go back into its historical records of  
10 inert ingredients that were present in registered  
11 products up until that time and they did a complete  
12 listing of all of those inerts.

13 Now, you should know that that database  
14 contains a lot of entries that are no longer accurate.  
15 For one reason or another, people sometimes fail to  
16 update -- sorry, people fail to remove from the EPA  
17 files confidential statements of formula that are no  
18 longer applicable to registered products. They have an  
19 obligation to file a new one. They don't also take the  
20 administrative step of saying: EPA, please pull the  
21 old one, get rid of it.

22 The other thing is that people --  
23 registrants, for whatever reason, tend to keep  
24 registrations active, even for products that aren't  
25 really being sold. So in going through that database,

1 EPA must have come up with inert ingredients that  
2 really are no larger in use for one reason or another.  
3 The reason I know this is because Environ Corporation  
4 provided support to EPA in the development of that  
5 inert ingredient policy and one of things we did was to  
6 help them discover, shall I say, the strengths and  
7 limitations of their own database for coming up with  
8 this list of inert ingredients.

19                           In putting this list together, EPA did  
20                           not -- in the exhibit that you have, EPA did not  
21                           designate the reason for listing any one of those  
22                           chemicals. Those reasons are available but in a  
23                           different document.

Now, List 2 consists of inert ingredients that have a high priority for testing. The reason they

1 have a high priority for testing is because some  
2 scientific concerns have been raised about potential  
3 toxicity or environmental effects. Many, if not most  
4 of the chemicals on List 2, have already been  
5 identified by the National Toxicology Program as  
6 canidates for testing.

7 List 4 is the list that contains  
8 ingredients that are of no concern. These ingredients  
9 are generally recognized as safe or grass status - that  
10 is conferred by the U.S. Food and Drug Administration -  
11 or they have been through some sort of review or  
12 they're exempted from the requirement of the tolerance,  
13 but in general there are no concerns about the list for  
14 chemicals.

15 And then there is List 3 which is  
16 essentially the other category, things that have not  
17 yes been categorized. This regulatory policy is  
18 intended to be fluid, as more information comes in  
19 chemicals may move from one list to another. It is  
20 fully expected that chemicals that are now on List 2  
21 will end up either on List 1 or List 4 as testing  
22 proceeds.

23 Now, for chemicals that are -- for inert  
24 ingredients that are on List 1, the EPA has established  
25 data requirements. There will be no new registrations,

1 new products registered containing those inerts unless  
2 people supply data to show that the presence of those  
3 inerts doesn't pose a hazard and there is an extensive  
4 list of data requirements that is being posed.

5                   For existing products, existing  
6 registrations that contain List 1 inerts, the agency  
7 required that the labelling of products containing  
8 those inerts carry a warning statement as of October  
9 1987 and that warning statement has to say something to  
10 the effect of: Warning, this product contains the  
11 toxic inert so and so.

12                  Now, the requirement to put that kind of  
13 a statement on a label is a strong incentive to the  
14 manufacturer to get that inert ingredient out of the  
15 formulation and that's for two reasons. First of all,  
16 because, as you know, in the United States the identity  
17 of inert ingredients is confidential business  
18 information, it is trade secret, the formula is a  
19 secret and the manufacturer doesn't like to divulge  
20 that.

21                  The second reason is that the appearance  
22 of a warning statement like that on a label quite  
23 rightly alarms consumers, purchasers, the users of the  
24 product and has negative impacts on sales, and EPA knew  
25 what it was doing when it imposed that labelling

1 requirement. The intent behind it was to get people to  
2 get those List 1 ingredients out of their formulations  
3 and to substitute things that were less toxic.

4 I won't go through the details of that  
5 exhibit since you already have it before you, but  
6 testing requirements have also been established for the  
7 List 2 ingredients. They will be dealt with on a  
8 case-by-case basis as they become before the agency  
9 and, as I said, this entire process will be ongoing.  
10 So as new information comes in, things will continue to  
11 change.

12 Q. I believe we are ready to move to the  
13 next overhead?

14 A. Yes. The next overhead, please.

15 MR. CASSIDY: That will be page 3 of  
16 Exhibit 1240, Madam Chair.

17 DR. RACHMAN: Now, as you know from  
18 previous testimony, I believe Dr. Ritter talked about  
19 this in detail, the United States EPA has specific  
20 protocols and guidelines established for the conduct  
21 and the reporting of the studies that are required for  
22 registration and these things determine whether or not  
23 the data that are developed are acceptable for  
24 reviewing the registration process.

25 The guidelines contain several parts.

1       First of all, they are referred to as EPA pesticide  
2       assessment guidelines and you will hear people  
3       abbreviate that as the guidelines. The first part of  
4       the guidelines consist of the standards and the  
5       protocols for the individual tests that are outlined in  
6       40 CFR, Part 158. Those protocols establish things  
7       like the number of animals that should be used, how the  
8       dosage should be selected and administered, how the  
9       animals should be housed and those sorts of things.

10                  In recent years, EPA has issued addenda  
11       to those guidelines which specify not only how the  
12       tests should be done, but how the results should be  
13       recorded. So that the test report itself has to be  
14       organized in a very specific manner, sections have to  
15       appear in a certain order, they have to be explained in  
16       a certain way and so on.

17                  Let's skip No. 3 here for a moment, I  
18       will come back to it. The last item, good laboratory  
19       practices. These are regulations that cover the way a  
20       test is performed and how the data are handled. These  
21       have applied to toxicology studies since 1978. Since  
22       October 1989, these regulations now apply to all  
23       testing done in any subject area for pesticide  
24       registration in the United States. These good  
25       laboratory practices or GLP requirements can be thought

1       of as kind of an institutionalization of standard good  
2       scientific practice. These prescribe procedures that  
3       any good scientific investigator working in a reputable  
4       laboratory would normally use in designing and  
5       executing a test; however, there are a few requirements  
6       that sort of go above and beyond what a good scientist  
7       would formally do and those requirements are in the  
8       areas of record keeping and retention, sample retention  
9       after the test is complete, those sorts of things.

10                   So what I am getting at here is that the  
11       fact that a test does not meet GLP standards is not  
12       necessarily an indication that it is not a  
13       scientifically valid test. It could be failing to meet  
14       some of those requirements for sample retention or  
15       record keeping that are really way beyond what a good  
16       scientist would normally do.

17                   Now, a failure to meet these criterion  
18       constitutes a data gap; that is, if a test is not done  
19       according to a standard accepted protocol or if it is  
20       not reported according to the standard report formats  
21       or if it was not performed according to the letter of  
22       the GLP regulations, that test is not acceptable for  
23       review by the agency. The agency won't even look at  
24       the scientific content of the study if it doesn't get  
25       past that first stage of review.

1 Point No. 3 here, the EPA standard  
2 evaluation procedures, are not properly part of the  
3 guidelines and perhaps I should not have put them on  
4 this slide. Those are actually standard operating  
5 procedures of EPA's that determine how an EPA reviewer  
6 should review particular studies. The reason I  
7 included it here is because potential registrants look  
8 at those SOPs, they contain very important information  
9 about what EPA is looking for in a study, and so it  
10 provides information to registrants when trying to  
11 design studies.

Okay. Now, as I said, data gap applies to cases in which a study does not meet these criteria. There is also another definition of data gap and that is a situation in which information is missing. That could arise for several reasons that I could think of. Commonly it comes up because the data requirement could not have been anticipated by the registrant at the time that the pesticide was registered.

20                           In the registration process, it often  
21                           happens that EPA requires some additional special study  
22                           that is not on the Part 158 list or which may be  
23                           indicated as conditional and the registrant is not  
24                           certain that that will be a requirement. In the  
25                           initial review of that package, the EPA may designate

1       that as a data gap. If it's not a serious data gap and  
2       the absence of that does not detail a significant  
3       adverse effect, EPA may go ahead and grant what's  
4       called a conditional registration. That means that the  
5       registration is granted, conditional upon the  
6       registrant providing that additional information at a  
7       certain point in the future.

8                 If the agency makes the determination  
9        that the absence of that data constitutes a potential  
10      for a significant adverse effect, they will not grant  
11      the conditional registration, they will wait until  
12      those data are supplied and they have a chance to  
13      evaluate them.

14                 Now, I would like to point out some  
15      additional things. Let's go to overhead No. 4, please.

16                 These are other requirements that apply  
17      to data developed for registration. I did not include  
18      this in the statement of evidence, but I would like to  
19      bring this to your attention. The agency has  
20      established what they call flagging criteria. The  
21      intention behind this is to prioritize for agency  
22      attention certain information that may indicate a  
23      potential for significant adverse effects.

24                 These requirements apply to any new  
25      information that is generated and submitted to EPA,

1       either in connection with a new registration or the  
2       support of an old registration, an update of the  
3       database. What these regulations require is that if  
4       the results of the studies at issue contain certain  
5       factors, certain results obtained, you must put a  
6       statement on the cover bringing to the agency's  
7       attention that dose effects have been noted and what  
8       that does is literally raise a red flag. When the  
9       agency receives that package those studies go into a  
10      priority review. The whole system is intended to allow  
11      the EPA to be extremely responsive to new information  
12      coming in that may have any significant -- that may  
13      have the potential to indicate adverse effects.

14                   These criteria apply to toxicology data  
15      and they apply to all the major areas of testing,  
16      oncogenicity or the cancer studies. I might point out  
17      that EPA uses the oncogenicity because they are  
18      concerned with not only malignant tumors, but also  
19      benign tumors.

20                   The chronic feeding studies and  
21      subchronic feeding studies, I believe you know about  
22      those, those are the two year or 90-day studies.  
23      Teratogenicity is birth defects. Neurotoxicity and  
24      reproduction I think are self explanatory.

25                   If we can have the next overhead, please.

1                           MR. CASSIDY: Q. I understand that this  
2 section of your evidence, Dr. Rachman, will deal with  
3 the post registration or requirements imposed by the  
4 EPA?

5                           DR. RACHMAN: A. That's correct. Once  
6 registration is obtained the registrant is not  
7 finished; there are more obligations that apply.

8                           There is a section in FIFRA in the law,  
9 section 6(a)(2), that specifies that any time  
10 information comes to a registrant on a registered  
11 product and that information has the potential to  
12 indicate adverse effects, the registrant must make EPA  
13 aware of that information within 15 working days of  
14 finding out about it.

15                          Now, there are regulations that have been  
16 developed that go into great detail about what kind of  
17 information must be recorded and what I have done on  
18 this overhead is to just summarize in a very general  
19 way the types of information that are covered by this  
20 requirement.

21                          MR. CASSIDY: And, Madam Chair, this  
22 evidence will relate to a issue raised in the scoping  
23 session by yourself dealing with the EPA registration  
24 process and its ability to make changes as scientific  
25 information becomes available.

1 Q. Go, ahead Dr. Rachman.

2 DR. RACHMAN: A. The registration  
3 relates to completed toxicity studies and they also  
4 cover certain incomplete toxicology studies. If you  
5 have a study that's ongoing and during the course of  
6 that study you notice certain things happening to the  
7 animals and test organisms that may signal the  
8 potential for adverse effects, you have to report that  
9 to the agency even before the test is completed, even  
10 before you have all the information in that allows you  
11 to determine whether or not that really is an adverse  
12 result.

13 Epidemiological studies are covered,  
14 including studies of efficacy of product. If a  
15 registrant begins to get information that his product  
16 is no longer effective for the purposes for which it  
17 was registered, that is a reportable incident. If  
18 residues in the diet or in the environment are found to  
19 exceed the levels that were anticipated during the  
20 registration process based on the registration data,  
21 that has to be reported to the agency. Also any  
22 incident that occurs, poisoning, et cetera that come to  
23 the registrant's attention. All that information has  
24 to be reported.

25 MADAM CHAIR: Excuse me, Mr. Rachman,

1       does that include -- does that focus more on the  
2       information that the registrant will obtain through the  
3       review of scientific literature as opposed to its own  
4       testing that it has done on its product?

5                     DR. RACHMAN: It includes --

6                     MADAM CHAIR: That's a wider net for a --

7                     DR. RACHMAN: That is exactly the term I  
8       was going to use. The regulation casts a very wide  
9       net, is doesn't restrict the registrant to look at one  
10      particular body of information, so it does include the  
11      tests that's are underway for whatever reason, the  
12      scientific literature, case reports from the medical  
13      literature, reports from various state agencies that  
14      monitor pest use and accidents and so on.

15                  I can conceive of cases, for example,  
16       where there might be a report in the media of an  
17       incident involving a pesticide use and my  
18       interpretation of the regulations would be that the  
19       registrant would have the responsibility to follow-up  
20       on that and determine whether or not that was a  
21       reportable result under Section 6(a)(2).

22                  MADAM CHAIR: And how long does that  
23       obligation continue for the registrant?

24                  DR. RACHMAN: Forever once the product is  
25       registered.

1                   MADAM CHAIR:  Forever.

2                   DR. RACHMAN:  Yes.  And compliance --

3        there are 15 work days for compliance once the  
4        information -- when the registrant becomes aware of the  
5        information and there are substantial fines for  
6        non-compliance.

7                   Another very important aspect of this  
8        whole procedure is that when the agency receives these  
9        class disclosures they are made public and reports of  
10      them appear in the Trade Press routinely.  By the Trade  
11      Press I mean publication of pesticide and toxic  
12      chemical use --

13                  Q.  I'm sorry?

14                  A.  Toxic chemical use published in the  
15      States.  And so this is a very rapid technique for  
16      making the public aware of adverse effect disclosures  
17      that have been filed.

18                  I think I would like to go on and talk  
19      about reregistration.

20                  MR. CASSIDY:  That overhead is No. 6,  
21      Madam Chair, of Exhibit 1240.

22                  Q.  I would ask you, Dr. Rachman, to  
23      describe first the history and authorization of the  
24      registration process and then the process itself.

25                  DR. RACHMAN:  A.  I'd just like to return

1 to the adverse effects for one moment, I neglected to  
2 make an important point which was implicit I think; and  
3 that is, the agency has the authority to take  
4 regulatory action at any time based upon that 6(a)(2)  
5 information that is submitted.

6 Q. What type of regulatory action are  
7 you referring to, Dr. Rachman?

8 A. Well, I will talk a little bit later  
9 about the remedies that are available to the agency in  
10 circumstances where risk is presumed.

11 MR. CASSIDY: And that will also relate  
12 to the scoping session issue raised by yourself, Madam  
13 Chair.

14 Q. If you could then move to the  
15 reregistration process which is described in the  
16 evidence and in the overhead No. 6, Exhibit 1240,  
17 please?

18 DR. RACHMAN: A. Yes. Now,  
19 reregistration has been required since 1972 when FIFRA  
20 was amended to incorporate that requirement and, in  
21 fact, in 1988 the law was amended and there is a whole  
22 new section of FIFRA now, Section 4, which deals  
23 extensively with reregistration requirements and  
24 procedures.

25 I would like to point out that I made an

1 error in my statement of evidence. I referred to FIFRA  
2 Section 3(g), this is on page 11 of my statement of  
3 evidence -- I referred to Section 3(g) of FIFRA, that  
4 was the old FIFRA. I should have put Section 4 because  
5 as of 1988 all of the registration requirements, I  
6 believe, have been moved to Section 4.

The 1988 amendments formalize the reregistration process, set out procedures and time lines. Very important. A nine-year deadline has been imposed by which time EPA must complete the reregistration of all active ingredients and they have been directed to prioritize this reregistration process based on the same sorts of potential exposure criteria that I mentioned before.

15 They were required to establish four  
16 lists of chemicals and associated deadlines for the  
17 submission of data and review of those data and so on.  
18 Those lists have been now been published. List A  
19 consists of active ingredients for which a registration  
20 standard has already been issued. Those chemcials are  
21 already in the process somewhere. There are  
22 approximately 192 of them.

23                           MADAM CHAIR: Excuse me. Is this  
24 separate from the four lists we just discussed?

25 DR. RACHMAN: Yes, Madam Chair. Those

1 lists refer to inert ingredients, reregistration of  
2 active ingredients, although I should point out that in  
3 reviewing the data on active ingredients and preparing  
4 the registration Standard, EPA also considers the  
5 formulated product. It is just that administratively  
6 it is all designated by the name of the active  
7 ingredient.

8 Let's see. So List A consists of the  
9 chemicals that -- they are already in the works. Lists  
10 B, C and D are the rest of them and the deadline for  
11 submitting the first information on List B chemicals  
12 has just passed, the deadline for the first information  
13 submission on List C chemicals is in July, and I  
14 believe the deadline for the List D chemicals is three  
15 months after that. So things are going to proceed at a  
16 very rapid clip. Nine years is not a long time, I  
17 should point out, for doing this kind of extensive  
18 review and I will get into that a little bit later.

19 The Congress directed EPA to prioritize  
20 these active ingredients for reregistration  
21 consideration using the following factors: whether or  
22 not they are used on food; whether or not they have  
23 significant outstanding data gaps; and whether or not  
24 there is significant potential for worker exposure.

25 So the chemicals that have the highest

1 ranking according to those three criteria are on List  
2 B, so they were the first ones to start the process;  
3 the next lower set of rankings is List C; and then the  
4 ones with the least concern in that area are in the  
5 List Ds.

6 Now, on this overhead, what I'm trying to  
7 provide to you is my own personal understanding of this  
8 process and how it works. There is at present,  
9 interestingly enough, no definition of reregistration  
10 and that's because, by its very nature, it is an  
11 ongoing cyclical process. I don't believe it was ever  
12 intended to have an ending

13 Reregistration was mandated in the first  
14 place because of the recognition that scientific  
15 advances eventually mean that data requirements change  
16 and protocol requirements change and as science  
17 continues to advance, data requirements will change to  
18 change. I am sure that years from now we will be  
19 testing pesticides for effects that we never even  
20 dreamed of testing for, simply because basic research  
21 will lead us in the direction of evaluating the  
22 concerns.

23 So what typically happens in the  
24 registration standard process is that EPA starts by  
25 taking a look at what's in the file, what's in the

1 database for the chemicals that's been registered and  
2 determining what data should be supplied to fulfill the  
3 registration requirements.

4 Now, that includes two sorts of  
5 considerations. One, are there no new data  
6 requirements today that did not apply when the chemical  
7 was first registered; and, secondly, are there any new  
8 protocols in place today, new guidelines that were not  
9 in place when the chmcial was first registered.

10 A deficiency in either of those areas;  
11 that is, either missing information or information that  
12 doesn't come up to the grade are current contemporary  
13 protocols, either deficiency is labelled a data gap in  
14 the registration standard process and you can't really  
15 tell from looking at the EPA documents what kind of  
16 data gap you are looking at.

17 You can appreciate, I am sure, that there  
18 is a vast difference in the quality of those two kinds  
19 of data gaps. In the one case, you have absolutely no  
20 information available to you as to whether or not you  
21 have got a potential adverse effect in the area that  
22 you are talking; in the other case, you do have  
23 information, but what you've got for some reason does  
24 not up come to current EPA standards.

25 Now, in fact there are some good examples

1       of that with some of the chemicals of interest to this  
2       proceeding, 2,4-D, for example. Dr. Ritter talked in  
3       his testimony about data that was available to Canada,  
4       that was not available to the EPA in the areas of -- I  
5       believe it was mutagenicity and metabolism.

6                     The EPA said that all of those studies  
7       were unacceptable. They were unacceptable because they  
8       didn't meet current EPA guidelines for the performance  
9       of those studies, and yet the data are of sufficient  
10      scientific quality that the expert panel conveyed by  
11      the Ministry of the Environment here in Ontario relied  
12      on them in assessing potential risks of 2,4-D.

13                  The way the EPA process works is that if  
14      the data don't pass that initial screen as to form, the  
15      scientific content is not even evaluated, they don't  
16      even look at it, and they designate in the registration  
17      standard that the information is not available, there  
18      is a data gap.

19                  Now, the EPA will issue a document that's  
20      called the registration standard, I have been referring  
21      to that. That's a document that lays out what the data  
22      requirements for the active ingredient should be -- I  
23      should say are, what the data requirements are, whether  
24      or not the EPA currently has information that satisfies  
25      those data gaps and -- I'm sorry, that satisfies those

1 data requirements and where data gaps are indicated  
2 deadlines, by which time new information must be  
3 submitted if the registration is to continue.

4 In going through all the existing data,  
5 the EPA evaluates the potential risks and if the data  
6 that are in the file, even if they are incomplete, if  
7 they should indicate that there exists a potential  
8 adverse effect, then the EPA has remedies available to  
9 it and it will often take interim action at that point,  
10 and I will talk about that in a moment.

11 When the new information comes in, and it  
12 may take a couple of years, depending on what kinds of  
13 tests are required and how long they take to run, there  
14 is what's called a second round review. The EPA  
15 re-evaluates the risk picture in light of new evidence,  
16 issues new findings as to the potential risk and  
17 sometimes requires more data.

18 Now, because it can take as much as four  
19 years for some of these tests to be done, particularly  
20 the chronic studies, and it can take a year or two for  
21 EPA to review the studies, in the meantime, as the  
22 guidelines for other studies have been updated, new  
23 data gaps can spring up. So you can see that this is  
24 as cyclical process. While you are filling one data  
25 gap another one may pop up, so then you have to go back

1 and review that study and that's just the way it was  
2 intendewd to be, this constant upgrading of the  
3 database.

4                   This is presenting some interesting  
5 problems for EPA now in light of this nine-year  
6 deadline that's been imposed by the 1988 amendments to  
7 FIFRA. A nine-year deadline implies that at the end of  
8 nine years they have to be at the end of something and  
9 they are right now wrestling with a definition, at what  
10 point are they going to be able to say to Congress that  
11 they have complied with that nine-year deadline when in  
12 fact they have a process that was intended to go on  
13 forever, and this should be very interesting to watch.  
14 There are high level meetings going on in Washington  
15 for the last couple of months trying to wrestle with  
16 this very important policy problem.

17                   Let me just remind you that in the  
18 development of this new information, anything that's  
19 required under a registration standard, those flagging  
20 criteria apply. So that if you are doing a new toxic  
21 study and you get a result that's indicated you have to  
22 bring it to the agency's attention immediately.

23                   The 6(a)(2) adverse effects disclosures  
24 also apply because you are dealing with a product  
25 that's already registered. So if you are doing a new

1       cancer study, for example, and you get some adverse  
2       effect that indicate a different order of toxicity than  
3       your old studies indicated, you may have to disclose  
4       that under the adverse effects disclosure.

5                   Again, this illustrates that EPA is --  
6       EPA procedures are set up so that they are responsive  
7       to incoming information that indicates potential for  
8       adverse effects.

9                   I would just like to remind you that the  
10      existence of a data gap does not imply a risk of any  
11      kind. I can't emphasize this point enough. Because of  
12      the different kinds of deficiencies that are all  
13      covered by that same term, 'data gap', you have to be  
14      very careful. If someone says to you there is a data  
15      gap on a chemical, it does not necessarily imply that  
16      there isn't any information there that you can use to  
17      evaluate whether or not there is a risk and it doesn't  
18      imply that evaluation could not be done. What it means  
19      is that the information may not meet EPA's criteria.

20                  The question you should ask always, I  
21      would suggest, is whether or not there is any  
22      information there at all that's useful in a scientific  
23      sense, whether the information is missing. Whether or  
24      not it is acceptable to EPA has no bearing practically  
25      on its scientific validity.

1                   Q. All right. I understand we are ready  
2 to move to the next overhead.

3                   A. I believe we are.

4                   Q. And this will be overhead No. 7 of  
5 Exhibit 1240.

6                   Madam Chair, this section will be -- I  
7 would like to ask Dr. Rachman to describe the EPA's  
8 authority to act when significant risks are identified  
9 and she will deal with the powers of the EPA once that  
10 occurs.

11                  DR. RACHMAN: Okay. I have tried to  
12 indicate how information that suggests potential  
13 adverse effects can come to EPA in a variety of ways  
14 and at any time, and now I would like to talk to you  
15 about some of the remedies that they have available to  
16 them when they decide there is a problem or potential  
17 problem.

18                  I have tried to organize these, just for  
19 discussion purposes, in ten -- in order of, shall we  
20 say, ease of EPA imposing this action. The amendment  
21 of terms of registration, for example, the first one is  
22 something that EPA can do at any time under the  
23 authority of FIFRA. Whenever they make the  
24 determination that there is some risk they can impose  
25 conditions on the registration; that is, changes on the

1 labelling, for example, requirements for protective  
2 clothing, something like that, that are intended to  
3 mitigate the risk and they routinely do this during the  
4 registration standard process while they are waiting  
5 for more information to come in that will allow them to  
6 do a more in-depth risk assessment.

7 For example, some of the chemicals at  
8 issue here are involved in the registration standard  
9 process. I can give you some examples. Now, let me  
10 point out that these risk reduction measures are  
11 specific to individual uses of the chemical, okay. The  
12 examples I am going to give you are not for forestry  
13 uses of these chemicals, although these are the  
14 chemicals that are approved for forestry use in  
15 Ontario.

16 The reason I will not give you examples  
17 for forestry uses is that we were unable to find any  
18 cases in which EPA has imposed risk reduction measures  
19 on forestry uses. There have been no risks identified  
20 specific to forestry uses; that is, human health risks  
21 and there have been no interim human health risk  
22 reduction measures imposed for forestry uses.

23 Now, with that said let me give you some  
24 examples. The fenitrothion registration standard,  
25 which was issued in 1988, imposed an interim 24 hour

1 re-enty interval for greenhouse uses of fenitrothion.  
2 This is because EPA felt that there was not enough  
3 information available in the files about the potential  
4 exposures of greenhouse workers using fenitrothion. So  
5 they imposed a data requirement on the registrant to do  
6 such a study and in the meantime, until those data are  
7 submitted and evaluated, there is a 24 re-enty interval  
8 which means that after a greenhouse is treated with  
9 fenitrothion workers cannot re-enter the premises for  
10 24 hours and signs have to be posted and so on.

11 I can also give you another example for  
12 glyphosate. Again, not a forestry related use. This  
13 one applies, I believe, to the agricultural and aquatic  
14 uses of glyphosate. In developing a registration  
15 standard, the EPA became aware of some incident reports  
16 from the State of California - California has a  
17 monitoring system in place - and apparently there were  
18 some reports of irritation and severe irritation,  
19 ocular and dermal, I believe, from uses of glyphosate  
20 by mixers and handlers and these incidents splashing of  
21 the formulated product and so on. So in this case, EPA  
22 imposed a label requirement for protective clothing of  
23 specifically face shields and so on to cut down on the  
24 probability of those kinds of worker exposures and  
25 accidents.

1                   I will review for you at the end of my  
2 talk the EPA status of the chemicals at issue here in  
3 this proceeding and I will come back to the interim  
4 risk reduction measures at that time and make  
5 corrections to overhead No. 11 in your packet.

6                   Okay. The next remedy available to EPA  
7 is special review, and I guess we could go to the next  
8 overhead at this point.

9                   Q. All right. That will be overhead  
10 No.8 of Exhibit 1240.

11                  A. Overhead No. 8. The process that's  
12 now called special review was was formally called  
13 rebuttable presumption against registration or RPAR,  
14 and thankfully EPA changed the name. RPAR was  
15 originally intended to evaluate the evidence to see  
16 whether unreasonable adverse effects are likely to  
17 occur. This is the procedural review of the evidence  
18 to make that determination.

19                  The original RPAR did not specify that  
20 EPA consider exposure data and the result I think was a  
21 very cumbersome process because every time there was  
22 some indication of inherent toxicity of a compound they  
23 would have to do this indepth evaluation.

24                  As you know from previous evidence, Dr.  
25 Ritter talked about this in detail, there is no risk if

1       there is no exposure. There may be toxicity, but if no  
2       one is exposed there is no risk. So in 1985 -- well,  
3       in 1978 FIFRA was amended to take this consideration  
4       into account, and in 1985 EPA formalized the new  
5       process and gave it the name special review to signify  
6       that there had been a change.

7                   There are specific criteria for the  
8       initiation of a special review and the criteria  
9       specified that the process can only be initiated if EPA  
10      has validated test evidence or other significant  
11      evidence raising prudent concerns of unreasonable  
12      adverse risk, and the word risk is in the regulations,  
13      and of course that implies not only toxicity but also  
14      exposure.

15                  What the special review process is, then,  
16      is a more indepth review of the evidence. If the  
17      agency makes a finding that there is evidence  
18      indicating a risk, they will begin the process of an  
19      indepth determination of whether unreasonable adverse  
20      effects occur and, as I showed on an earlier slide,  
21      unreasonable adverse effects includes not only risk,  
22      but also benefit. So you can think of the special  
23      review process as an indepth risk benefit evaluation of  
24      the use of the pesticide.

25                  Now, this slide here, No. 8, shows you

1 the initiation criteria in the area of human health  
2 effects. There are also criteria for environmental  
3 problems and I have not dealt with those since our  
4 evidence is dealing with human health effects.

5 Again, the standard of the evidence is  
6 validated test or other significant evidence. I would  
7 point out that EPA has to include not only the active  
8 ingredient in its consideration, but also impurities,  
9 metabolites, and so on. The criteria you can see here  
10 are very broad, a wide range of effects could indicate  
11 a concern provided that the evidence is of sufficient  
12 quality the agency can initiate a special review.

13 Special reviews are rare and the reason  
14 that they are rare is because the agency has to have a  
15 high quality of evidence in order to be able to  
16 initiate one. They have to be able to make the  
17 determination that there is the potential for  
18 unreasonable adverse effect for a risk, and frequently  
19 the evidence that's available just does not allow them  
20 to make that determination. We have provided in our  
21 statement of evidence EPA's latest position with  
22 respect to 2,4-D.

23 Q. And I understand you wish to update  
24 that with respect to what has happened since?

25 A. Yes, I will do that. Can I wait

1 until we get to slide No. 11?

2 Q. Certainly.

3 A. I should say that this is not the  
4 latest position. This was the latest position at the  
5 time we prepared our statement of evidence. I will  
6 update you at the end of our talk.

7 Q. Yes.

8 A. In this notice, EPA announces that it  
9 has decided not initiate the special review on 2,4-D,  
10 and to paraphrase liberally, the reason is that the  
11 evidence just does not indicate that there is a risk of  
12 carcinogenicity.

13 Q. All right. The next overhead?

14 A. Let me just check here for a moment.  
15 I would just like to say a word about the procedures of  
16 special review because some of those terms may come up.  
17 Yes, let's go to overhead No. 9.

18 Q. This is overhead No. 9 in Exhibit  
19 1240.

20 A. The EPA special review process is  
21 like the registration standard process, lengthy.  
22 Probably it would be fair to say that a special review  
23 cannot be concluded in under two years except under  
24 what I would say unusual circumstances. The  
25 presumption starting a special review is usually that

1 there is a risk that has been identified that may be so  
2 serious that the registration has to be cancelled.  
3 That's usually the story, and then the registrant has  
4 the burden of providing evidence to EPA to show that  
5 the risk not that great.

6 EPA usually imposes data requirements and  
7 asks for specific information that it would like to see  
8 in order to do a better determination of the potential  
9 risk.

10 MADAM CHAIR: Excuse me. Is that after  
11 they cancel the registration or--

12 DR. RACHMAN: No, it is not.

13 MADAM CHAIR: --it would follow the  
14 cancellation until they...

15 DR. RACHMAN: Yes. They may make an  
16 initial proposal that cancellation might be required,  
17 but they can't proceed to cancellation until they have  
18 made the final determination that there does exist an  
19 unreasonable adverse effect and that there is no way to  
20 mitigate it except to cancel the registration.

21 MADAM CHAIR: And they can't do that  
22 usually under two years?

23 DR. RACHMAN: That's my impression, Madam  
24 Chair. I have not done a thorough study of all the  
25 special reviews that have been done, but I have been

1 involved with a couple on specific chemicals and my  
2 recollection is that the procedure took in the order of  
3 two to three years to come to its regulatory  
4 resolution, which was not always cancellation, and I  
5 will talk about that.

6 MADAM CHAIR: And in the meantime in that  
7 two year period when possible cancellation and the  
8 reason for it is being studied, does EPA put on special  
9 conditions to mitigate that as well?

10 DR. RACHMAN: They may do so, yes,  
11 exactly. The initiation notice kind of puts the  
12 registrant on notice that EPA has identified a certain  
13 potential risk and kind of, you know, casts the net for  
14 information and then also advises the registrant of  
15 particular data requirements and deadlines for  
16 supplying the data.

17 The position documents, there have been  
18 as many as four that I am aware of, sequential  
19 documents, within the course of a special review. Each  
20 one is an update of the agency's position vis-a-vis the  
21 risk and the proposed regulatory measures in light of  
22 evidence that has come in since the last position  
23 document.

24 This is a very important point, that the  
25 standard of evidence here in a special review

1 proceeding is very high and so the agency cannot move  
2 quickly through this process. These are generally very  
3 difficult scientific questions that are dealt with and  
4 there is a lot of interpretation, a lot of  
5 scientific -- back and forth and until there is a  
6 resolution and a final risk assessment that people are  
7 happy with, it takes some time.

8 Now, the outcome -- let me just mention  
9 the scientific reviews which is point No. 3. Section  
10 25(d) of FIFRA directs that whenever EPA is proposing  
11 the cancellation of a registration they have to submit  
12 that proposal and the scientific rationale to a  
13 scientific advisory panel, and the section goes on to  
14 give criteria for selection of panel members and so on.

15 Basically these are outside independent  
16 scientists with recognized credentials and a lot of  
17 experience and they serve limited terms on the panel  
18 and they advise the agency about proposed  
19 cancellations. The agency also makes liberal use of  
20 the SAP to evaluate proposed regulatory policies and  
21 other scientific issues that arise in the course of  
22 registration. They meet regularly.

23 So this outside peer review is a part of  
24 this process and frequently the SAP will review an  
25 agency proposal and say that they don't agree with the

1 agency's position, the evidence doesn't support it,  
2 they need more information and so on, and that  
3 frequently is one reason why the process takes so long,  
4 because these scientists direct that other things  
5 should be considered, other data should be developed  
6 and so on. The SAP role is advisory; EPA does not have  
7 to listen to what they say, but in practice its  
8 suggestions are generally taken into account.

9 Now, the outcome of a special review is  
10 not a regulatory action, it is a recommendation for an  
11 action. The outcome of a special review is that  
12 weighing of risk versus benefit for a particular use,  
13 the use that's at issue or for all uses, depending on  
14 what the problem that is identified is. If that  
15 risk/benefit relationship cannot be brought into  
16 acceptable balancing, then cancellation is an option.

17 Could we go to the next overhead, please.

18 Q. Overhead No. 10, Exhibit 1240.

19 A. These are the two regulatory options  
20 that EPA has available once a significant risk has been  
21 identified, the final determination of risk has been  
22 made. Cancellation requires, as I've said,  
23 unreasonable adverse effects have been identified and  
24 the project -- I'm sorry, the product does not meet the  
25 registration requirements that I illustrated on my

1 first slide.

2 Now, the thing about cancellation is that  
3 the law and regulations provide that effected parties  
4 may ask for a hearing which, of course, they almost  
5 always do and those hearings are quite lengthy and the  
6 cancellation process can, therefore, take in the order  
7 of a couple of years.

8 MADAM CHAIR: Excuse me. A hearing  
9 before whom?

10 DR. RACHMAN: It is an administrative  
11 hearing.

12 MADAM CHAIR: Before --

13 DR. RACHMAN: Before an administrative  
14 law judge on the question of unreasonable adverse  
15 effects and agency procedures in evaluating those  
16 risks.

17 While this is going on or even during a  
18 special review, registrants may choose to voluntarily  
19 cancel their own registrations. You will occassionally  
20 hear of this happening. Frequently -- well, I  
21 shouldn't say frequently. My impression of why this  
22 happens is that in some cases the registrant may  
23 recognize that there is in fact a significant risk and  
24 that there is no point in going through a cancellation  
25 proceeding because ultimately the product will be

1       cancelled anyway.

2                   There is another situation that occurs in  
3       the United States and that is, because all of these  
4       proceedings were public and there is a tremendous  
5       public concern about pesticides, as there is here in  
6       Canada, adverse publicity or publicity about adverse  
7       effects has a deleterious effect on sales. So in some  
8       cases manufacturers may just make the decision that  
9       selling the product is no longer feasible, but for  
10      whatever reason, it is possible for a manufacturer to  
11      voluntary cancel at which point the process just ends.

12                  If there is a special review going on,  
13       EPA simply publishes a notice terminating the special  
14       review. If there is a cancellation proceeding in place  
15       they just terminate that and that's it, cancellation is  
16       effective.

17                  Okay. So the cancellation proceeding  
18       takes some time. Now, in going into a cancellation  
19       proceeding, EPA will do an analysis of how much risk,  
20       if you will, will attain during the period it's going  
21       to take to cancel this product and if they decide that  
22       that's an unacceptable risk, let's say two more years  
23       of use of this product or 18 months or whatever is  
24       going to cause an unacceptable risk, instead of  
25       cancellation they have the option of suspension.

1                   In order to propose suspension they have  
2 to find -- make a finding of imminent hazard. Imminent  
3 hazard is what I just explained. It's a finding that  
4 during the time it would take to cancel this product  
5 the risk would be unacceptable.

6                   Now, registrants have the option of  
7 requesting a hearing on suspensions as well, even  
8 though suspensions are effective within 30 days of the  
9 notice. If the registrant requests a hearing, they  
10 have to have a hearing and that may stretch the  
11 proceeding out to several months.

12                  If EPA decides that during that period of  
13 several months, the time needed for a suspension  
14 hearing, the risk would be unacceptable, they have a  
15 further option, they can do an emergency cancellation  
16 which is -- I'm sorry, an emergency suspension which is  
17 effective immediately. No hearing.

18                  This is a very, very rarely exercised  
19 option, but in fact it has been exercised recently and  
20 that was the emergency suspension of dinoseb. Dinoseb  
21 is not used for forestry. A registration standard was  
22 issued on dinoseb in 1984, it has a variety of  
23 agricultural uses. As a result of that registration  
24 standard, the registrant had to do a new teratology  
25 study, a birth defect study, and when the results of

1       that study were submitted to EPA, the EPA determined  
2       that the margin of safety for handlers and applicators,  
3       mixer, loaders, and so on, people handling dinoseb, the  
4       margin of safety was unacceptable.

5                     Because of where they were during the  
6       calendar year, the dinoseb use season was upon them and  
7       the agency made the determination that the number of  
8       people that would be at risk from the use of dinoseb  
9       within the coming six months was unacceptable and under  
10      those conditions they moved for an emergency  
11      cancellation of dinoseb which became effective -- I  
12      think it was 1985. I would have to check that for you.

13                    MR. CASSIDY: I think we are ready to  
14      move on to the next section and this section of Dr.  
15      Rachman's evidence, Madam Chair, deals with the EPA  
16      status of pesticides approved for forestry use --

17                    MADAM CHAIR: Excuse me, Mr. Cassidy.

18                    MR. CASSIDY: Yes.

19                    MADAM CHAIR: Just one question for Dr.  
20      Rachman. Is it an emergency suspension or emergency  
21      cancellation?

22                    DR. RACHMAN: Emergency suspension, Madam  
23      Chair.

24                    MR. CASSIDY: Sorry, Madam Chair.

25                    The next section deals with the EPA

1       status of pesticides approved for forestry use in  
2       Ontario and we will be referring to a table which can  
3       be found in the witness statement at page 21, which is  
4       now on an overhead, and as Dr. Rachman indicated, she  
5       would like to make a slight correction to the table  
6       which she will indicate to you now.

7                     DR. RACHMAN: Shall we do the correction  
8       first?

9                     MR. CASSIDY: Whatever is best for you.

10                  DR. RACHMAN: Well, since we have already  
11       discussed it I might as well refer to those...

12                  MR. CASSIDY: This is Table 1, Madam  
13       Chair.

14                  DR. RACHMAN: The correction I wish to  
15       make is in the right-hand column under fenitrothion. I  
16       would like to insert the example that I gave you  
17       earlier in my testimony about the interim re-entry  
18       requirement imposed for greenhouse and nursery uses of  
19       fenitrothion pending submission of exposure data.

20                  MR. CASSIDY: I am indicatig right on the  
21       right-hand corner for your benefit, Madam Chair, where  
22       that change should be made of Table 1.

23                  Q. Dr. Rachman, if you could just speak  
24       up just a bit so that the people at the back of the  
25       room can can hear you.

1 DR. RACHMAN: A. Certainly.

2 Q. Thank you.

3 A. Would you like me to repeat that?

4 Q. Yes, I think Ms. Seaborn needs that  
5 again.

6 A. What I would like to have entered  
7 there is the fact that an interim re-enty interval of  
8 24 hours for greenhouse and nursery uses of  
9 fenitrothion was imposed pending the receipt of  
10 exposure data.

11 Q. That is, again, a non-forestry use;  
12 is that correct?

13 A. That's correct.

14 MADAM CHAIR: What year was that, please?

15 DR. RACHMAN: I will have to check on  
16 that for you, Madam Chair. Well, the registration  
17 standard was 1987.

18 MR. CASTRILLI: Excuse me, Madam Chair,  
19 may I ask the witness to repeat the very last portion  
20 of that comment. It was pending receipt of...?  
21 I didn't catch the remainder.

22 DR. RACHMAN: Exposure data. The  
23 agency's concern was that available information was not  
24 sufficient to determine whether greenhouse and nursery  
25 workers were at risk, so they asked for exposure data

1 to be developed and until such time as they can  
2 evaluate it, they imposed this interim re-entry  
3 interval.

4 Now, let me emphasize that our review of  
5 the available information showed that no human health  
6 risks have been identified with respect to forestry  
7 uses of the chemical pesticides approved for use in  
8 Ontario and, therefore, no -- there have been no  
9 cancellation proposals, no suspensions and no interim  
10 risk reduction measures have been imposed.

11 I felt, though, that that would make a  
12 pretty boring chart if it was all nos, so what I did  
13 was to include, just for purposes of your information,  
14 some of the risk reduction measures that have been  
15 imposed for other uses so that you can see the kinds of  
16 things that the EPA has done for some of these  
17 chemicals.

18 Before we start, let's just clarify once  
19 more the registration status of a couple of these  
20 chemicals. First of all, aminocarb or Matacil is no  
21 longer registered in the United States. There were no  
22 regulatory actions against it, as far as we could  
23 determine. My conclusion is that it was a voluntary  
24 cancellation on the part of the registrant and I do not  
25 know why. I would expect it had to do with some market

1 consideration, but I really can't say for sure.

2 Fenitrothion is not registered for food  
3 use. It is registered for forestry uses, it is also  
4 registered for use on -- I'm afraid I can't remember  
5 the exact terminology, it is domestic and commercial  
6 and industrial insecticidal use, so it's registered for  
7 use around the home for insecticidal purposes, but not  
8 on foods or in food handling areas.

9 All of the other chemicals are registered  
10 for food uses. So that means that in putting together  
11 the registration standards, EPA is evaluating these  
12 chemicals with respect to the most stringent and  
13 extensive data requirements of 40 CFR, Part 158. The  
14 registration amendments column applies to uses other  
15 than forestry uses since there have been no actions  
16 against the forestry uses.

17 Now, we can take these in turn. 2,4-D.

18 When we prepared our statement of evidence, this  
19 special reviews that had been proposed in 1986, EPA  
20 decided in March of 1988 that the available evidence  
21 just did not warrant the initiation of a special  
22 review.

23 Since we prepared this statement of  
24 evidence, we became aware of a subsequent notice  
25 published in the Federal Register in October of 1989

1 and in that notice EPA reserves the right to initiate a  
2 special review and announces that it will postpone  
3 making the decision as to whether or not a special  
4 review is warranted until it receives two epidemiology  
5 studies which were currently underway at that time and  
6 which it had become aware of.

7 MR. CASSIDY: Q. And I understand you  
8 wish to file a copy of that notice?

9 DR. RACHMAN: A. Yes.

10 MR. CASSIDY: This would be exhibit, I  
11 believe, 1242, Madam chair, entitled A Notice of Status  
12 of Consideration for a Special Review. (handed)

13 MADAM CHAIR: Thank you.

14 ----EXHIBIT NO. 1242: Document entitled Notice:  
15 Status of Consideration for a  
Special Review.

16 DR. RACHMAN: Now, that notice which has  
17 just been filed as an exhibit sets out some deadlines  
18 by which the agency expected to receive these studies.  
19 To the best of my knowledge, those studies are still  
20 not available as of last week, in fact that was what  
21 Environ was able to determine.

22 Now, may I ask see a copy of that notice?

23 MR. CASSIDY: Certainly.

24 DR. RACHMAN: Thank you. Let me just  
25 make sure that my...

1                   On the last page of the notice EPA says  
2                   that they are waiting for two NCI, National Cancer  
3                   Institute, case control studies. The first study from  
4                   eastern Nebraska has been completed but results will  
5                   not be released to EPA until the end of 1989. As far  
6                   as I know those results have still not been released.

7                   Then the notice goes on to say:

8                   "Statistical analysis of the second  
9                   study, from Iowa and Minnesota, is in  
10                  progress, with release anticipated in  
11                  March of 1990."

12                  We were unable to determine whether that  
13                  study has been released yet. We think it has not.

14                  So EPA is going to postpone its decision  
15                  as to whether or not to initiate a special review until  
16                  such time as it has an opportunity to review those  
17                  studies.

18                  MADAM CHAIR: Excuse me, let me get this  
19                  straight. In the chart that we are looking at, the  
20                  special review proposed in 1986, a decision was made...

21                  DR. RACHMAN: Not initiate in 1988.

22                  MADAM CHAIR: And then in 1989 they  
23                  decided they would -- they might initiate a special  
24                  review depending on --

25                  DR. RACHMAN: That's correct. What they

1 are saying essentially is that we've heard that there  
2 is some new evidence on the way, this evidence may in  
3 fact trigger a special review, we can't tell until we  
4 see it, we will let you know when we see it, whether  
5 the evidence supports the initiation of the special  
6 review or not.

7 MR. CASSIDY: And that is the effect of  
8 Exhibit 1242?

9 MADAM CHAIR: That's correct..

10 MADAM CHAIR: So what was added to Table  
11 1 in the witness panel, Exhibit 1240?

12 DR. RACHMAN: That's correct.

13 MADAM CHAIR: Okay.

14 DR. RACHMAN: Now, we should point out,  
15 there are also some animal studies relating to the  
16 carcinogenicity of 2,4-D that are outstanding and yet  
17 to be completed.

18 However, my interpretation of this notice  
19 is that the EPA is not going to wait to receive the  
20 data from these animal studies in order to make the  
21 decision as to whether or not to initiate the special  
22 review, that decision will be made based on these  
23 epidemiology studies which should be available well in  
24 advance of the animal data.

25 Now, just let me belabor this point a

1 little bit more. What we are talking about here is the  
2 initiation of a special review of a risk benefit  
3 determination, not a cancellation. And I hope I've  
4 made it clear in my testimony the relationship between  
5 the two, and the fact that what the agency is talking  
6 about initiating for 2,4-D is an indepth review and  
7 would probably take several years to conclude.

A. "In making the determination...."

15 Q. Yes.

16 A. It says:

22 The word is missing, but...

Q. I think it is "during".

1 comprehensive evaluation."

2 Q. All right. Thank you.

3                           MADAM CHAIR: So they are not putting  
4                           into place any conditions with respect to the  
5                           registration?

6 DR. RACHMAN: No, that's correct. That's  
7 correct.

8 MR. CASSIDY: I believe that concluded  
9 yours evidence, Dr. Rachman --

10 DR. RACHMAN: In fact, it does not.

11 MR. CASSIDY: I'm sorry.

12 DR. RACHMAN: I would like to say a few  
13 more things. I will be brief.

14 MR. CASSIDY: We are breaking at 10:10;  
15 is that correct, Madam Chair?

16 DR. RACHMAN: I will be finished before  
17 that, I promise.

21 DR. RACHMAN: Really, I have almost ran  
22 out of steam here.

I think the other entries on this table  
are self-explanatory. The only other chemical  
pesticide that has ever been even considered for

1       special review is carbaryl. That was way back in the  
2       late 1970s and there again in 1980 the agency published  
3       a notice that the available evidence just did not  
4       support the initiation of special review.

5                   I do not recall what the issue was at  
6       that point. There was a toxicity issue, but I'm sorry,  
7       I just don't remember what the details were.

8                   MADAM CHAIR: So the initiation of this  
9       special review of the risk/benefits of 2,4-D is  
10      applicable only to the agricultural use of the product,  
11      not to a forestry use of 2,4-D, or obviously if they  
12      find some risk in the agricultural side--

13                  DR. RACHMAN: That's right.

14                  MADAM CHAIR: --it would extend to other  
15      uses.

16                  DR. RACHMAN: If the question is the  
17      toxicity of the material, it is in the case of 2,4-D,  
18      they will examine all the uses and all the exposures to  
19      see what the risks are under every condition of  
20      exposure that's allowed by the current registration, so  
21      the forestry use will certainly be considered.

22                  MR. MARTEL: They would allow use of a  
23      product in forestry simply because of the infrequency  
24      at which it would be used as opposed agriculture?

25                  DR. RACHMAN: Well, that might turn out

1 to be the case, Mr. Martel.

2 MR. MARTEL: I am not saying they are  
3 constant, just based on the frequency of the  
4 application of it?

5 DR. RACHMAN: Right. Dr. Rodricks will  
6 be talking about these sorts of issues in his  
7 testimony. When you are looking at cancer risk, the  
8 extent of exposure over a lifetime is a very important  
9 variable.

10 So you are absolutely right, they would  
11 make take a look at the extent of lifetime exposure and  
12 make the determination of lifetime cancer risk, and it  
13 might happen that the cancer risks for certain uses,  
14 perhaps for forestry, would be within an acceptable  
15 level, whereas the risks of certain other uses, perhaps  
16 the agricultural uses, would not. That is the domain  
17 of risk assessment and that's what Dr. Rodricks will be  
18 talking about.

19 I would like to point out one other thing  
20 which is not on this chart but just for your  
21 information. The EPA maintains a categorization system  
22 for chemicals that are thought to be carcinogenic and  
23 its what's called a weight of evidence classification  
24 and it's a convenient ranking scheme to express the  
25 nature and the quality of the evidence of

1       carcinogenicity on those various chemicals.

2                     Now, being on this list implies nothing  
3       about risk, we are talking strictly about the inherent  
4       toxicity of a chemical, strictly the evidence that it  
5       causes cancer. Whether or not it causes a risk under  
6       use conditions is, again, a subject for risk  
7       assessment, it depends on the exposure under use  
8       conditions of a chemical. So what we have here is  
9       just -- it's a classification scheme.

10                  Since we prepared our statement of  
11       evidence, two of the chemicals of interest here have  
12       changed classification and I'm not going to go into any  
13       great detail on the scheme, it will probably be more  
14       appropriate for Dr. Rodricks to do deal with that  
15       later, but I will just explain the two changes that  
16       have occurred.

17                  MR. CASSIDY: Q. And I understand you  
18       wish to file a document which outlines those two  
19       classifications?

20                  A. Yes, that's correct.

21                  MR. CASSIDY: This will be the next  
22       exhibit, Exhibit 1243, Madam Chair, and if it can be  
23       titled EPA Updates List of Classified Carcinogenic  
24       Pesticides. That title you will see at the very -- on  
25       the front of Exhibit 1243.

---EXHIBIT NO. 1243: Document entitled EPA Updates List of Classified Carcinogenic Pesticides.

DR. RACHMAN: Now, this exhibit is not an official agency publication, it is a reprint of an article I found in Pesticide and Toxic Chemical News, which is the trade publication I mentioned before.

Apparently someone in Congress requested an update from EPA as to the carcinogen classification of currently registered active ingredients and EPA sent this list over to Congress and it was reported in the press. EPA will eventually issue an official memorandum. They occassionally issues these lists, not on a regular basis.

MR. CASSIDY: Perhaps we can note for the record that this update, Exhibit 12423, is dated May 2nd, 1990.

DR. RACHMAN: Now, the two items of interest here are 2,4-D -- I'm sorry, there are three, 2,4-D, glyphosate and simazine.

2,4-D and glyphosate have moved to the D category. The D category is the one that essentially means we don't have enough information to tell whether or not this is a carcinogen or not. We have required new studies, we are waiting for the results.

Simazine is listed as category C.

1       Category C is the one that entails limited evidence of  
2       carcinogenicity. This means that there were tests  
3       performed in two species, rat and mouse. This simazine  
4       was positive in only one, only in rat and is not  
5       carcinogenic in the mouse, and I believe category C  
6       also implies that the material is not a genotoxic.

7                   Is that correct, Dr. Rodricks?

8                   DR. RODRICKS: Yes.

9                   DR. RACHMAN: Does not cause mutations in  
10       mutagenicity studies. So the designation for chemicals  
11       in category C is possible human carcinogen which  
12       reflects uncertainty as to whether the animal evidence  
13       indicates that it is a potential human carcinogen or  
14       not. I just wanted to bring those to your attention.

15                  MADAM CHAIR: Now, to clarify this, a  
16       congressman has asked that this be updated?

17                  DR. RACHMAN: Yes.

18                  MADAM CHAIR: And has EPA done --  
19       reclassified these?

20                  DR. RACHMAN: Well, they have -- yes,  
21       they have updated their list. They maintain a list of  
22       some kind and there is an ongoing evaluation process.  
23       As new data come in on various chemicals in the  
24       registration standard process, these categories may be  
25       changed to reflect the latest data and this list

1 doesn't appear with any regular frequency, it is not a  
2 mandated list, but usually once or twice a year they  
3 update to reflect new categories -- or changes in  
4 categories that have been assigned.

5 I might also point out that the science  
6 advisory panel gets involved in reviewing these  
7 qualifications. So the EPA will propose a  
8 classification for a chemical based on its review of  
9 new studies and present that to the SAP and the SAP  
10 will, you know, invite comment from the public and  
11 evaluate the information and either concur or disagree  
12 with the agency classification.

13 MR. CASSIDY: All right. Just one final  
14 matter to be dealt with before the break, Madam Chair.

15 The evidence refers to -- the witness  
16 statement refers to the farm mortality study which, you  
17 may recall, was also discussed by Dr. Ritter in his  
18 evidence back in MNR's Panels 12 and 13. You may  
19 recall that at the time Dr. Ritter gave his evidence  
20 and indeed at the time this witness statement was  
21 written, the farm mortality study was only available in  
22 an abstract form.

23 It is referred to on page 62 of the  
24 witness statement, but since the publication of this  
25 witness statement and the evidence of Dr. Ritter, that

1 study has now become available and just before we break  
2 I would like to provide you and the parties with a copy  
3 of it and it will be discussed by Dr. Rodricks briefly  
4 in his evidence, and if that could be Exhibit 1244,  
5 being the Saskatchewan Farm Mortality Study. I have  
6 paraphrased the title because it would take the length  
7 of the break to read the title, so I will just call it  
8 that.

9 MADAM CHAIR: What's the date on that?

10 MR. CASSIDY: It is dated April 4th,  
11 1990.

12 MADAM CHAIR: Who are the authors?

13 MR. CASSIDY: The authors are Wigle et  
14 al. (handed)

15 MADAM CHAIR: Thank you.

16 ---EXHIBIT NO. 1244: Document entitled Mortality Study  
17 of Canadian Male Farm Operators:  
18 Non-Hodgkin's Lymphoma Mortality  
and Agricultural Practices in  
Saskatchewan.

19 MADAM CHAIR: Please remind me, for whom  
20 was this study done? Was it for the National Cancer  
21 Institute or the Federal Government in Ottawa?

22 MR. CASSIDY: It was --

23 DR. RACHMAN: I am afraid I don't  
24 remember.

25 MR. CASSIDY: It was carried out -- Dr.

1       Ritter was one of the participants. It is a Canadian  
2       study and I believe was carried out through the  
3       Laboratory Centre for Disease Control, although I am  
4       not certain of that, Madam Chair, and it may be helpful  
5       to go back and review the evidence of Dr. Ritter.

6                     MADAM CHAIR: Okay, thank you.

7                     MR. CASSIDY: It is a Canada-wide study.  
8       However, Saskatchewan was, as you will recall from Dr.  
9       Ritter's evidence, and as is referred to in the  
10      abstract, the first province in Canada for which data  
11      was produced.

12                  It might be appropriate now for the  
13      break, Madam Chair, and we will commence after it with  
14      Dr. Rodricks' evidence.

15                  MADAM CHAIR: Perhaps you could have the  
16      witnesses just review the study quickly over the break  
17      and let us know for whom it was done. That will save  
18      me going back to looking at the Ritter evidence.

19                  DR. RACHMAN: Certainly.

20                  MR. CASSIDY: All right.

21                  MADAM CHAIR: Thank you, Mr. Cassidy.

22                  The board will be back in 20 minutes.

23      ---Recess taken at 10:15 a.m.

24      ---On resuming at 10:45 a.m.

25                  MADAM CHAIR: Please be seated.

1                   Mr. Cassidy, just a short announcement.  
2                   We have a new telephone number and new fax number if  
3                   the parties want to take this down. Our telephone  
4                   number here at 151 Bloor Street is 963-1249. That's  
5                   963-1249, and the new fax number (416) 963-1252.

6                   MR. CASSIDY: Does that fax come into  
7                   these premises, Madam Chair?

8                   MADAM CHAIR: Yes.

9                   MR. CASSIDY: Thank you. Madam Chair,  
10                  does the Board intend to announce today the schedule  
11                  for Panel 10, the OFIA panel?

12                  MADAM CHAIR: Later today or tomorrow  
13                  morning.

14                  MR. CASSIDY: Thank you.

15                  MR. FREIDIN: Madam Chair, just on that  
16                  point. I can advise that if the Board wishes to  
17                  commence a week earlier than the 13th that poses no  
18                  problem for the Ministry.

19                  MADAM CHAIR: Thank you, Mr. Freidin.

20                  MR. CASSIDY: Madam Chair, we are now  
21                  prepared to commence with the second section of this  
22                  evidence and the second witness, Dr. Rodricks, who is  
23                  before you. For your notes, this section commences at  
24                  page 24 of the witness statement, Exhibit 1239, through  
25                  to the conclusion of the evidence, page 73.

1                   Dr. Rodricks will be referring to a  
2                   number of overheads, which have been filed as Exhibit  
3                   1241, in the course of the presentation of his  
4                   evidence. I will turn on the first overhead which can  
5                   be found at page 1 and Dr. Rodricks will commence by  
6                   discussing the various approaches to health risk  
7                   evaluation which are outlined on that overhead and in  
8                   the evidence.

9                   DR. RODRICKS: Thank you, Madam Chair.  
10                  We were asked to discuss general approaches now taken  
11                  for the evaluation of human health risks from  
12                  substances in the environment, and to define some of  
13                  the terms of that evaluation, at least as they are used  
14                  in the United States, and then to look more  
15                  specifically at some evaluations that have been  
16                  reported from various groups on pesticides used in  
17                  forest settings.

18                  So I am going to proceed, then, through  
19                  each of those steps by beginning with a general  
20                  discussion of the process of risk assessment as it's  
21                  called, or risk evaluation as I call it here, and I  
22                  think it is important to do this. This may be quite  
23                  familiar to many of you, but I find that there is  
24                  sometimes confusion with respect to certain terms and  
25                  their use in the process.

1                   I might also say that the terms that I am  
2                   going to describe here came out of a review conducted  
3                   by the National Academy of Sciences in the United  
4                   States, 1983, a review of what had been going on in the  
5                   federal government with respect to the evaluation of  
6                   risks from chemicals in the environment.

7                   The academy committee that looked at the  
8                   issues said that there is a consistency in approach,  
9                   there has been for quite a long time, but not a good  
10                  consistency in the use of terms and they proposed a  
11                  specific way in which risk assessors in government and  
12                  outside of government ought to organize information and  
13                  to have it proceed through the evaluation process, and  
14                  the committee noted that whether one was talking about  
15                  a chemical risk or any other kind of risk, that there  
16                  is a general procedure, a way to think about the  
17                  problem and to organize your evidence.

18                  So this procedure, which includes a  
19                  four-step process which I will go through here, is now,  
20                  I would say, used, at least in the United States, in  
21                  almost every situation where individual -- where either  
22                  governments or other individuals are looking at the  
23                  question of potential risk.

24                  I might say that in other areas of the  
25                  world, I am familiar with what what goes on in this

1 kind of evaluation, there are some differences among  
2 countries in the use of certain terms and there are  
3 even some analytic differences in the way certain  
4 problems are approached and I will point out one of  
5 those, but generally you will find that in any  
6 evaluation these same steps are followed. They may be  
7 called other things and may not appear, as I said, in  
8 this stepwise fashion, but it is the general approach  
9 toxicologists follow.

10                   The first step of the process, what we  
11 call hazard identification, concerns the issue -- first  
12 of all, by hazards we mean any inherent dangerous  
13 property, in this case the chemical agent. Some agents  
14 are highly flammable, some chemicals are radioactive,  
15 we are particularly concerned with the issue of  
16 toxicity. The first step in evaluation is to survey  
17 scientific -- after you have identified the agent in  
18 which you are interested, to survey the scientific  
19 literature and attempt to identify --

20                   MADAM CHAIR: Excuse me, Dr. Rodricks,  
21 can we just have a short break.

22                   MR. CASSIDY: They have to report a  
23 spill.

24                   MADAM CHAIR: I did the same thing  
25 myself, Mr. Castrilli, this morning.

1                   MR. CASTRILLI: Madam Chair, let the  
2 record show that I was not the source of the spill, I  
3 was simply the victim of the spill.

4                   MR. CASSIDY: I think the source of the  
5 spill was a representative of the Ministry of the  
6 Environment.

7                   ---Discussion off the record

8                   MR. CASSIDY: Just while we are waiting,  
9 it might be an opportunity for Dr. Rachman to advise  
10 the Board with respect to the last question on the farm  
11 mortality study. We have pulled the transcript of Dr.  
12 Ritter's evidence and I think Dr. Rachman can interpret  
13 it for the benefit of the Board.

14                  DR. RACHMAN: Madam Chair, do I  
15 understand your question correctly that you were  
16 interested in the purpose for conducting the study?

17                  MADAM CHAIR: No, who funded the study?  
18 Was it --

19                  DR. RACHMAN: Well, I'm not sure I can  
20 answer that particular question, but I will read to you  
21 from Dr. Ritter's testimony. This is Volume 122, page  
22 20,463. The question to Dr. Ritter from Eleanor Cronk  
23 was:

24                  "Who was The author of this abstract?"  
25 His answer is:

1                   "There were five of us. These are the  
2                   joint collaborators on this study which  
3                   are drawn from the Centre for Disease  
4                   Control, the Laboratory Centre for  
5                   Disease Control of the Department of  
6                   Health and Welfare and the Environmental  
7                   Health Directorate, my group of the  
8                   Department of Health and Welfare."

9                   And that's really all he says. I don't  
10                  see anything here specific to the funding of this  
11                  study.

12                  MADAM CHAIR: That's fine. Thank you.

13                  MR. CASSIDY: Q. I believe we are now in  
14                  a position to proceed Dr. Rodricks.

15                  DR. RODRICKS: A. I was just beginning  
16                  to go through quickly the four steps of any complete  
17                  risk evaluation.

18                  The first step, as I said, concerns  
19                  identification of the agent you are interested in, in  
20                  our case, for example, 2,4-D or other herbicides, and  
21                  to survey the scientific literature to identify the  
22                  kinds of toxicity, the specific agent seems capable of  
23                  causing under some conditions. All chemicals will  
24                  cause toxicity under some conditions, the important  
25                  point is what type of toxicity, does it cause cancer,

1       does it cause birth defects, does it effect  
2       reproduction, does it irritate the skin, and those  
3       properties of chemicals vary widely among them. So you  
4       want to find out whether a chemical can cause - the  
5       important word there is cause - specific kinds of  
6       conditions under some conditions.

7                  Now, the primary sources of information  
8       for this, among toxicologists and risk assessors, come  
9       from studies in experimental animals. The reason for  
10      why resort to the use of experimental animals even  
11      though that's not the rodent -- laboratory rodents are  
12      not the species of interest to us ultimately, is that  
13      these are studies that have two pretty, I should say,  
14      characteristics that make them very, very important.

15                 No. 1, you can gather information on  
16       toxicity before you permit human exposure to take  
17       place. That's very, very important. No. 2, if animal  
18       studies are well done, it is possible, relative easy  
19       and possible to establish causation in a fairly  
20       straightforward way; that is, if you do the experiment  
21       well, you have animals that receive the agent of  
22       interest at various levels and then a set of control  
23       animals that are identical in every single respect  
24       except they do not receive the agent of interest, so if  
25       there is an effect you can attribute causation fairly

1       easily.

2                     Then the third important point is,  
3     although it is not perfect, we believe animal results  
4     are applicable to people with some exceptions and some  
5     qualifications, but there is enough basis for believing  
6     that if you see a carcinogenic response in animals that  
7     is convincing, there is a reason to be concerned that  
8     the agent may also present a cancer hazard to people.  
9     Again, that's not a perfect -- that's not perfect  
10    knowledge, but we behave as if that's the case  
11    generally.

12                  Epidemiology studies - and I will come  
13    back to this point in a little more detail later - are  
14    obviously of great importance because they are studies  
15    in human beings. The problem is that, No. 1, these  
16    studies are not controlled studies in any sense of the  
17    word like a laboratory study and it is a very, very  
18    difficult to establish a truly something approaching in  
19    a truly controlled situation. I will come back to that  
20    point a little bit later.

21                  Q. The farm mortality study is an  
22    epidemiology study; is that correct?

23                  A. Yes, and there are, as I will show  
24    later, 15 to 20 epidemiology studies on, if not 2,4-D  
25    itself, at least the broad class of herbicides.

1                    Anyway, it's very difficult -- the point  
2                    I was just going to make is that it's very difficult to  
3                    establish causation from epidemiology studies because  
4                    you don't have a truly controlled situation. You are  
5                    taking advantage of a situation where people are  
6                    already exposed and trying to set up something like a  
7                    controlled study, but it's not really achievable with  
8                    any single study.

9                    I might say that for chemicals in  
10                  general, most of the information that is the basis for  
11                  regulation, certainly for pesticides and for almost all  
12                  other chemicals, comes from animal studies. There are  
13                  hundreds of epidemiology studies in the literature on  
14                  chemical agents mostly in occupational settings where  
15                  you can have more intense exposures and a lot of those  
16                  show some kind of association between exposure and some  
17                  health conditions or cancer even, but there are only  
18                  about 30 where 30 chemicals or substances, mixtures of  
19                  chemicals where the evidence has arisen to the level of  
20                  what we would call a causal relationship, and I will  
21                  come back to that point later.

22                  At any rate, you need to look at both  
23                  kinds of studies when you are looking at a particular  
24                  chemical and make then a judgment about whether a  
25                  causal link exists. The second step, which I will deal

1 with more briefly now because I have a chart to  
2 illustrate this one in a couple of overheads later, we  
3 are trying to understand the relationship between  
4 exposure, the magnitude of exposure to the chemical,  
5 what we call the dose in sort of technical jargon, and  
6 the frequency and severity of adverse health effects  
7 because one of the principles of toxicology that not --  
8 the dose, the size of the dose determines how much risk  
9 there is for any agent, and I will come back to that  
10 point later, but we are trying to understand that  
11 relationship for each of the toxic effects of concern.

12                 The third step, then, is what is called  
13 the human exposure evaluation. The first two are  
14 specific to the chemical and come out of the scientific  
15 literature. The third step concerns the specific uses  
16 of the chemical that you might be interested in. In  
17 our case, the pesticides used in forest settings, we  
18 would like to know that given the conditions of use of  
19 those chemicals, who might come into contact with them,  
20 how much of those chemicals get into peoples' bodies or  
21 onto their bodies, how much of the chemical over what  
22 period of time. We would like to know the answers to  
23 all of those questions because all of these determines  
24 how much exposure, how much risk might arise.

25                 I hope you can see at this point that the

1       fourth step, what I have called -- the Academy of  
2       Science has called the risk characterization step  
3       involves integration of information from those first  
4       three. There is no new information here in this step,  
5       it's rather putting together all of the information in  
6       the first three steps: What kind of health effects,  
7       how do they relate to exposure, what is the actual  
8       extent of exposure, and putting that together you  
9       arrive at some picture of the risk, the likelihood that  
10      under specific conditions those harmful effects are  
11      going to appear. That is called the risk.

12                  I put down likelihood here and I will  
13      show you a minute that how likelihood is expressed in  
14      kind of qualitative terms, kind of a judgment, and it  
15      might be expressed in more quantitative terms as well,  
16      and I go into this in a little more detail on my second  
17      chart.

18                  One final point here, that these four  
19      steps comprise the risk assessment or risk evaluation  
20      process. They don't in themselves lead to a decision,  
21      but the use of a particular chemical -- there is a  
22      separate step in the process which we refer to as the  
23      risk management step that involves policy, the question  
24      of -- a decision about whether the risks are low enough  
25      to be considered insignificant or are they too high,

1       are they a significant public health burden and then  
2       other factors may also enter into a decision. As Dr.  
3       Rachman pointed out in connection with FIFRA in the  
4       U.S., benefits of the use of the pesticide are  
5       considered, et cetera, but those are a separate step  
6       and the risk analyst really doesn't answer those  
7       questions for us, although there are a lot of  
8       precedents that we can point out to for decisions about  
9       acceptable or significant or insignificant health risks  
10      based on this analysis.

11                   Let me then move on to the second chart.  
12          The Board asked during the scoping session some  
13          questions about weight of evid -- so-called weight of  
14          evidence evaluations or judgments versus more  
15          quantitative evaluations of risk and I want to address  
16          that briefly and the process.

17                   Q. Looking at page 2 of Exhibit 1241.

18                   A. Page 2. I do not see these as  
19          either/or kinds of situations, but rather I believe a  
20          full evaluation of risk deals with both what are sort  
21          of qualitative weight of evidence evaluations, as well  
22          as more qualitative evaluations.

23                   Step one of the process, the hazard  
24          evaluation, is largely a weight of evidence evaluation.  
25          It is not easy to quantify this; we are answering the

1 question: How likely is it that agent "x" causes birth  
2 defects and if we have animal evidence on that, we need  
3 then to judge the quality of all of the evidence  
4 available, we may then also look at human evidence and  
5 make an overall weight of evidence evaluation on the  
6 hazard question. So it's clearly a very important part  
7 of that step.

8                   The Ministry of the Environment panel of  
9 experts report on 2,4-D that I will come back to in a  
10 moment, is largely a weight of evidence evaluation on  
11 the question: Is 2,4-D a likely human carcinogen under  
12 any conditions. It is only the first step of the  
13 hazard -- of the risk assessment process, it is only an  
14 attempt to answer that first step, although the panel  
15 also got into some risk issues as well, and I will come  
16 back to that, but most of the report is this weighted  
17 evidence kind of evaluation.

18                   MR. CASSIDY: That MOE report is Exhibit  
19 714, Madam Chair.

20                   DR. RODRICKS: Steps 2 and 3, the  
21 so-called does response relationship and human exposure  
22 evaluation, we try to make those as quantitative as we  
23 can. There are some sort of qualitative judgments in  
24 that process as well, but it is very hard to reach firm  
25 conclusion without some quantitative information; how

1 much exposure do people have, that's more than a  
2 qualitative question. You try to get some quantitative  
3 information about the size of that. So that figure is  
4 quite important.

5 Step 4, the final risk characterization.  
6 I might say that the National Academy of Science panel  
7 chose the term risk characterization very carefully  
8 because they were concerned that given our state of  
9 knowledge these judgments couldn't be strictly  
10 quantitative in nature if we don't know enough, and  
11 that there ought to be accompanying any quantitative  
12 evaluation a kind of weight of evidence evaluation as  
13 well. A qualitative discussion of how good the  
14 evidence is and the uncertainties is not quantitative.  
15 So I see these as not either/or kinds of evaluations,  
16 but as really one in the same.

17 Now, I would also have to point out, and  
18 this comes up a great deal in the Crump evaluation as I  
19 will refer to it and that's Exhibit...

20 Q. 716.

21 A. There is uncertainty in any risk  
22 evaluation. I have never seen one in all my years of  
23 doing this that didn't have some uncertainties in it.  
24 There are two kinds. You may not always have all the  
25 data you want on a specific chemical or its uses, and

1       then there are also -- there are just some gaps in our  
2       basic scientific knowledge on many of these questions,  
3       that's another uncertainty. I will show you in the  
4       next graph a very, very important example of that.

5                     The general approach in the risk  
6       evaluation process is where you have a range of  
7       possibilities, scientific possibilities to choose from  
8       and you really can't pin down at a particular step in  
9       the analysis one or the other as better supported  
10      scientifically. The typical procedure in the risk  
11     assessment process is to choose that assumption which  
12     will yield the highest estimate of risk, that is called  
13     a conservative or worst-case kind of analysis.

14                  Now, you don't have to do that  
15     exclusively, we also like to give what is called a more  
16     typical kind of assumption or what we call a more mid  
17     range kind of assumption so that the decision-maker can  
18     look at what is perhaps a more reasonable kind of  
19     approach, but then you also want to look at what is the  
20     worst, the most pessimistic view of what might happen.  
21     The Crump analysis consistently uses, in at least one  
22     of their analysis, a series of worst-case assumptions  
23     that I will illustrate to go throughout the process  
24     where you have certain kinds of gaps in knowledge.

25                  The result of that is that - and I will

1 show a specific example in the next graph that I said -  
2 we can't claim that the risks we produce are  
3 quantitative estimates in particular, are accurate  
4 portrayals of human risk. We don't have any way to  
5 know that, at least from most exposures that occur in  
6 the environment, but we can say - and this is EPA  
7 language in the U.S. and I think quite widely  
8 accepted - that by using this procedure we put what is  
9 called an upper limit on the risk, an upper limit on  
10 how bad the risk might be. It could be accurate but we  
11 have no way to check it.

12                 The actual risk is most likely less than  
13 we're predicting by these methods and it could be zero,  
14 but we don't know that either. Again, we can't have  
15 perfect confidence, but high confidence that the risks  
16 are like to be upper bounds.

17                 If you look at the Board's review, they  
18 made a point that you can't quantify human risk and I  
19 would agree with that. We don't have the knowledge to  
20 do that, but we can quantify some upper limit on what  
21 it might be and we can be much more confident of that.

22                 Let me give you one example of that on  
23 the next chart which deals with this very tricky issue  
24 of dose response evaluations.

25                 Q. At page 2 --

1                   A. A little sense of what goes on in the  
2 process. This is a graph which plots risk, which I  
3 have over near on the vertical axis, and the units  
4 here -- let me just point to the units. With risks we  
5 are dealing with probabilities. Remember I said in the  
6 first chart it's the likelihood of some adverse effect,  
7 that is the probability here. Probabilities range from  
8 zero to one, they don't have any units. So we talked  
9 about a one in ten chance of something happening or a  
10 one in two chance or a one in a million chance or  
11 whatever.

12                  So I plotted over here on this axis the  
13 figure 0.1, that's a one in ten chance of something  
14 adverse happening, the risk is an adverse event, up to  
15 something like 0.9, then I drew a line across here at  
16 .1. I did that because -- and then I called this, the  
17 region from risks of .1 and higher, the range of  
18 observation and I will explain that in a second.

19                  The other axis, the horizontal axis, is  
20 the dose of the chemical, the exposure to the chemical  
21 or dose, more technically, the amount that gets into  
22 the body. If you are talking about -- this could apply  
23 either to animal studies or to situations where people  
24 are exposed to the chemicals.

25                  All our techniques for discovering

1       adverse effects are limited to quite high risk  
2       situations. It is just, as a statistical matter, very  
3       difficult or impossible to detect risks below about one  
4       ten, that's a pretty high risk, but with animal  
5       studies, unless you are going to use million and  
6       millions of test animals or with human studies, human  
7       studied tend to be even less sensitive, you have a  
8       limit of the risk you could detect. You just run out  
9       of -- it's like an analytical chemistry method, you  
10      just run out of statistical power to detect a risk.

11                   So I have called this the range of  
12       observation and one of the reasons in the animal tests  
13       you use very high doses, you use very high doses, much,  
14       much higher than people would ever be exposed to, to  
15       see if a risk can be produced and you do that because  
16       you are limited to detecting very, very high risks.

17                   So you will have -- I drew a straight  
18       line here as the sort of dose response relationship and  
19       I called that the observed response. I don't mean to  
20       imply that every observed response is a straight line,  
21       most of them are not, so this is kind of hypothetical,  
22       but that's what you typically observe mostly from  
23       animal studies.

24                   I would also add that in human studies,  
25       and it is a problem with every single one of the

1 studies we have on phenoxy herbicides in human studies,  
2 we really do not have any quantitative dose information  
3 from those studies at all. As a matter of fact, one of  
4 the serious problems is that we are not even sure which  
5 chemicals are involved in most of those studies. I  
6 will come back to that.

7 So I am not talking here about a specific  
8 chemical, but the general process because later in my  
9 discussion I shall be referring to some of the terms  
10 that come out in this discussion.

11 Now, if we look at most chemicals that  
12 enter the environment and the pesticides at issue here  
13 would be good examples, actual human exposures will  
14 tend to be rather low relative to those where you can  
15 observe effects. They will be way down in this range  
16 and that's quite typical. You may identify the few  
17 occupational situations where exposures may be high,  
18 but most typically environmental exposures are very,  
19 very much lower.

20 So the question is: If I can detect  
21 risks at very high doses and very high risks, then is  
22 there a risk and what is its size at very low doses,  
23 and that gets you into what is called extrapolation.

24 The general thinking now among  
25 toxicologists is that -- first of all, this so-called

1       dose response relationship isn't just going to end  
2       right here, it is going to continue below that even  
3       though you can't detect it. Then the question is: In  
4       what shape does it take in this range of extrapolation.

5                   There is strong evidence that for most  
6       toxic effects this will fall off at some point to what  
7       is called a threshold, a no-effect level, a threshold  
8       dose for most toxic effects, this is quite widely  
9       accepted, such that the risk really drops off to zero,  
10      at some dose well above zero and you can determine that  
11      dose experimentally.

12                  In risk assessment now, this assumption  
13       of a so-called threshold is now used everywhere that I  
14       know of for all kinds of toxic effects except cancer,  
15       and the general procedure here, the risk assessment  
16       procedure, is to identify that no-effect level  
17       experimentally and then ensure that there is some  
18       margin of safety as the term is called, margin of  
19       safety; people are protected from that range where you  
20       get effects by a margin of safety and there are some  
21       precedents for margins of safety for various kinds of  
22       toxic effects, and I will come to those.

23                  Cancer is more controversial. From the  
24       1940s and later, there is some experimental work for  
25       some substances capable of causing cancer; namely, the

1       radiation of various types, that this curve -- this  
2       relationship between dose and response came down in  
3       what is called a straight line or a linear relationship  
4       and the risk didn't really disappear until you got to  
5       absolutely no exposure.

6                   In other words, there was some risk all  
7       the way down under this straight line here, but did  
8       fall off. The probability of cancer would decline. So  
9       it is really -- as I said, the experimental evidence of  
10      for that is very limited and mostly to radiation,  
11      Certain kinds of radiation.

12                  There is some evidence for certain kinds  
13       of radiation that there might not be a threshold; that  
14       is, the risk goes to zero only when the dose -- at an  
15       absolute zero risk only when the goes to zero, but  
16       falls off in a curve line like this so that the curve  
17       line -- if that were accurate at a given dose, you see  
18       the curve line shows less risk than the straight line.  
19       That's true all the way up.

20                  Then there is also evidence for some  
21       agents that even carcinogens may follow a threshold  
22       kind of phenomenon. Again, this is an area of  
23       uncertainty. The general approach, unless you have  
24       very compelling evidence otherwise, is to assume that  
25       all carcinogens, whether they are animal carcinogens or

1 known to be human carcinogens, follow this straight  
2 line relationship.

3 I know of no evidence, I know of no  
4 analysis which suggests the risk could be higher than  
5 that straight line; that seems very, very improbable.  
6 There is substantial evidence that it could be less and  
7 it could be zero, but the general approach in risk  
8 assessment, unless you have some data on sort of a  
9 chemical specific basis, is to use the linear, no  
10 threshold model, so it's the most pesimistic of what  
11 could happen and this indeed what Crump did in his  
12 analysis and this is indeed what the Ministry of  
13 Environment expert panel did in their analysis.

14 I don't disagree with that, we just have  
15 to be careful interpreting the results.

16 Notice that -- one final point here or  
17 actually two small points. No. 1, when you follow the  
18 straight line analysis for a carcinogen -- I said for  
19 non-carcinogens what the risk assessment usually  
20 reports is the margin of safety; that is, the  
21 difference between the human exposure and the minimum  
22 toxic dose or the agent.

23 For carcinogens, what is reported when  
24 you adopt this quantitative procedure, this straight  
25 line, is to report -- let's say, you determine the

1       lifetime dose - this is really the lifetime dose we are  
2       talking about here - to be at some point along here,  
3       you then use the procedure to go up to the line and  
4       report the risk corresponding to that dose. So you  
5       will see in Dr. Crump's report and the MOE report  
6       presentations of risk as probabilities, mostly very,  
7       very low probabilities, done by that basic or that sort  
8       of analysis. So the risk is this probability of one in  
9       ten million, one in a million, et cetera, et cetera, et  
10      cetera.

11                  As I said, there are precedents to turn  
12       to to decide at what point you are going to say this  
13       risk is really negligible, there is no real need as a  
14       public health matter to worry about it. There are a  
15       lot of precedents, at least in the United States, for  
16       that, but that really is a separate risk management  
17       decision.

18                  The final point on this is that this  
19       linear, no threshold model and this quantitative  
20       expression of risk is used almost everywhere in  
21       regulation in the United States for carcinogens as a  
22       guide to regulation. As far as I know, it has not been  
23       widely used at least in other countries, in Europe, in  
24       Japan, in Canada. I know it has been discussed and  
25       there have been some examples of its use. I'm not sure

1 why that is because in many cases more weight of  
2 evidence kinds of judgments -- this threshold kind of  
3 approach might be seen as kind of a weight of evidence  
4 judgment and is used for carcinogens. Both of them  
5 have merits and both of them have weaknesses, but in  
6 the United States this procedure -- I feel fairly  
7 confident it's a meaningful procedure as long as you  
8 are careful to note what you are doing and the  
9 uncertainties in it, that you are not really predicting  
10 actual risk, there is really no way to know that.

11 Let me move on to the two specific issues  
12 that we are going to go over in a little more detail  
13 now, that is the general background and the context for  
14 dealing with them.

15 Q. That is Exhibit 3 of Exhibit 1241.

16 A. As I said before, we were asked to  
17 examine primarily an expert panel report of the  
18 Ministry of Health, dated March 23rd, 1987 which  
19 undertook a review on the question of the  
20 carcinogenicity of 2,4-D specifically.

21 Then we were also asked to look at a very  
22 extensive document that was prepared by Dr. Kenny Crump  
23 and his associates. Dr. Crump has a small firm in the  
24 Unite States, very much like our own, and they do  
25 similar kinds of analysis. We had never seen this

1 report before when we were asked to review its content  
2 to see if it conforms to good practices and risk  
3 assessment. So I have some opinions on each of those.

4 I might just emphasize, as I tried to  
5 before, that in the weight of evidence concerning the  
6 carcinogenicity of 2,4-D we are talking primarily about  
7 the question of whether it is -- whether it is known to  
8 cause cancer under any conditions, the hazard step, not  
9 the question of: Does it pose a risk, let's say, in  
10 forest settings. That's a separate question that  
11 requires -- if you decide in the first case that it  
12 does cause cancer, then you would proceed with the  
13 other steps to see how big the risk might be.

14 No. 4. I guess I need to add here, I  
15 guess it would be the appropriate point, that since our  
16 statement of evidence was prepared a second review of  
17 the weight of evidence to carcinogenicity of 2,4-D has  
18 been prepared by a group at Harvard University.

19 MR. CASSIDY: I have a copy of that  
20 document to file. It is titled the Weight of the  
21 Evidence of the Human Carcinogenicity of 2,4-D. It  
22 will be Exhibit 1245.

23 ---EXHIBIT NO. 1245: Document entitled The Weight of  
24 the Evidence of the Human  
25 Carcinogenicity of 2,4-D.

1 DR. RODRICKS: This report appeared in  
2 January 1990 and it was put together by Dr. John Graham  
3 at the Harvard School of Public Health. He convened a  
4 panel of experts in epidemiology and toxicology to look  
5 at the question of the carcinogenicity of 2,4-D, the  
6 hazard evaluation. So I have added this and some  
7 conclusions from that report here.

8 Both of these groups looked at three  
9 kinds of evidence and these are typically the kinds of  
10 things you would look at in making this evaluation, the  
11 available human epidemiology data, the available animal  
12 evidence, then I noted here on this chart other  
13 relevant data and by that I note -- and if you go  
14 through these reports you will see that the experts  
15 considered other information besides animal tests and  
16 human tests as part of the evaluation. They looked to  
17 see whether the material had any properties, for  
18 example, to indicate it could damage genes or cells.

19 That is one characteristic, it is not  
20 a -- whether it does nor not is not an overwhelming  
21 determinant of whether it is a carcinogen, but it is an  
22 important part of the evidence. They look at chemical  
23 structure, other toxicology data besides the cancer  
24 studies are relevant. So there was that kind of  
25 analysis in each of these reports. Each of the two

1 evaluations looked at those three kinds of evidence in  
2 great detail.

3 On the question -- on the human  
4 epidemiology questions, I am going to repeat a couple  
5 of points I made and go into them in a little more  
6 detail on the general criteria for evaluating  
7 epidemiology studies because, as I said before -- we  
8 will go to overhead No. 5.

9 As I said before, these studies are not  
10 controlled studies in the way a laboratory animal study  
11 is controlled. So judging whether a causal  
12 relationship - that's the ultimate question, do the  
13 studies show a causal relationship, that's very, very  
14 important - requires more than the usual kind of  
15 analysis, and this is a source of great confusion.

16 There are, depending on how you count  
17 them, 15 to 20 epidemiology studies reported since  
18 19 -- most of them appearing in the last decade. On  
19 phenoxy herbicides or -- that is the whole class of  
20 about a dozen different phenoxy herbicides that have  
21 been in use of which 2,4-D is one, and even herbicides  
22 more generally, pesticides more generally or other  
23 situations where exposures with these herbicides might  
24 be involved. 15 to 20 such studies. That is the body  
25 of evidence that these reports deal with.

1                   I said already that these are not  
2 controlled studies, but to varying degrees  
3 epidemiologists are able to, by taking proper  
4 precautions, set up something like a controlled  
5 investigation where you are comparing individuals with  
6 a disease to individuals without a disease and then  
7 comparing the differences in their exposures or,  
8 conversely, you might look at groups that have the same  
9 kinds of exposures and follow them to see whether --  
10 I'm sorry, you look at groups that have different  
11 exposures, some to herbicides and some not to  
12 herbicides, to see whether there is any different in  
13 the disease pattern in those groups.

14                  Those are the sorts of studies that are  
15 undertaken and you try to set up something like a  
16 controlled situation, but it is very, very rarely  
17 achieved and achieved a true control situation in none  
18 of these studies.

19                  Now, another thing that is very important  
20 to consider, and I am citing here some basic principles  
21 that are I think widely accepted by epidemiologists  
22 and, as far as I can tell, were used to guide the  
23 evaluation of the Ministry of Environment group and the  
24 Harvard group as well.

25                  First of all, the appearance of what is

1       called a statistical association between exposure and a  
2       health effect in a single study is not sufficient to  
3       confirm a cause/effect relationship. There are  
4       hundreds of epidemiology studies on pesticides and  
5       many other chemicals where you see associations; that  
6       is, in a given study there seem to be some excess of  
7       the rate of a certain disease, certain cancers, certain  
8       kinds of cancers and exposure to a particular chemical  
9       or chemicals. For many, many reasons, just that single  
10      observation would not be enough to establish causation.

11                  I can use a simple example of why that  
12        might be the cause. There is, for example, a strong  
13        association between the number of hours people spend  
14        watching television and the risk of coronary disease,  
15        but we didn't really believe -- that's a very strong  
16        statistical association, that televisions might cause  
17        coronary heart disease, but it is more likely that  
18        hours spent catching television reflects some other  
19        factor, maybe reduced physical activity that may be  
20        more directly related to it.

21                  So that sort of association gives you  
22        some clues and you can't ignore it, but, at the same  
23        time, you can't jump to a conclusion of causation.  
24        That's what we are trying to get to here.

25                  And you see that, you see in the studies

1       on phenoxy herbicides, several examples in the  
2       literature of associations between phenoxy herbicide  
3       exposure and certain kinds of cancers. I could list  
4       them. The earliest reports identified three kinds of  
5       cancers as possibly associated with exposure to  
6       phenoxyherbicides - these were reports from Sweden - of  
7       a group of cancers called soft tissue sarcomas, STS,  
8       and then two kinds of cancers of lymph cells.

9                   I was going to write them down here but I  
10          guess...

11                   Soft tissue sarcomas, these are just  
12          cancers of connective tissue of one type or another.  
13          .sarcoma is just one type of malignant cancer, and then  
14          what is called Hodgkin's disease, which is a cancer of  
15          certain lymph cells. It is a lymphoma, if you like,  
16          and then non-Hodgkin's lymphoma, and pathologists make  
17          a distinction between certain kinds of cells involved.  
18          in Hodgkin's disease as cancers of the lymph glands,  
19          lymph nodes and cells, and non-Hodgkin's lymphoma which  
20          is basically all other kinds of -- I'm not enough of a  
21          pathologist to distinguish, you need a pathologist to  
22          distinguish them, but they do distinguish these as  
23          different kinds of cancer that shouldn't be grouped  
24          together.

25                   Anyway, there were association reports

1       between exposures in Sweden among forestry workers,  
2       people involved in spraying right-of-ways with phenoxy  
3       herbicides back in -- these studies covered the period  
4       79, '80 and '81 and these three kinds of cancer  
5       associations were reported. That, of course, gave rise  
6       to a series of studies over the next ten years and they  
7       are still going on on these questions.

8                  Now, because of the problem in any  
9       particular study of distinguishing an association that  
10      the statistician might see, any true causal  
11      relationships, the epidemiologists seek - I have these  
12      five criteria listed under Item No. 4 here - generally  
13      seek before reaching a judgment about causation the  
14      following kinds of evidence.

15                 First of all, they look for consistency,  
16      a consistent pattern of associations in several  
17      studies, several undefined. There is no specific  
18      criteria here, but you would like to see it in several  
19      studies conducted in different populations by different  
20      methods of study, and I will go back and review where  
21      we are on phenoxies with respect to this criteria.

22                 B, if you want to establish a causal  
23      relationship, you ought to know what it is that those  
24      people were exposed to in as much detail as you can;  
25      what the chemical was, and how how much exposure

1 occurred and over what period of time. That's 4B.

2 C -- A and B are probably the strongest  
3 components of the analysis. C is nice to have,  
4 sometimes very difficult to apply and our  
5 epidemiologists realize this, that just as in my dose  
6 response relationship I showed you later -- I'm sorry,  
7 earlier, you would like to see evidence that as  
8 exposure increased in those populations the risk, the  
9 numbers of cases, if you like, of the cancer also  
10 increased.

11 There are many examples of instances  
12 where you see an association between exposure and an  
13 excess of cancer in one group, but then you look at a  
14 group that is more intensely exposed for a longer  
15 period of time and there is no association there. That  
16 would raise great doubt. You wouldn't say there isn't  
17 a causal relationship, but that sort of observation  
18 would raise great doubt about whether there is a causal  
19 relationship.

20 D, just the strength of the statistical  
21 association is important here. How strong the  
22 statisticians say this association is and then,  
23 finally, some evidence from animal studies. This would  
24 not be required, but if in fact you had confirmatory  
25 evidence from animals, that would add further weight to

1           the evidence.

2                         Now, these are general criteria and the  
3                         general procedure for reaching conclusions on any  
4                         specific case is to gather a group of epidemiologists  
5                         who look at all the available evidence and make a  
6                         judgment. This is not a simple formula that they might  
7                         carry through, they make a judgment about the weight of  
8                         that evidence and how closely or not so closely they  
9                         match these criteria.

10                  In the case of 2,4-D in particular, I  
11                 will go to 5(a) which lists the conclusions and I will  
12                 tell you a few of the reasons why both the MOE experts,  
13                 Exhibit 714, and the Harvard panel of experts reached  
14                 the conclusions they did.

15                  Q. That's Exhibit 1245, the Harvard  
16                 panel of experts.

17                  A. This is only on the human  
18                 epidemiology evidence, it's not the total evidence. I  
19                 will talk a little bit again about animal evidence and  
20                 other kinds of effects.

21                  With respect to the human evidence, the  
22                 MOE concludes, and I will read this quote and comment a  
23                 little bit about it and how it got there.

24                  "Using IARC terminology..." Let me  
25                 clarify that. IARC stands for the International Agency

1 for Research on Cancer. It is a unit of the world  
2 health organization in France and one of their -- they  
3 do cancer research, but one other thing they do that's  
4 very, very important is convene panels of experts to  
5 review cancer data on environmental agents and other  
6 agents as well, drugs and so forth, and reach  
7 conclusions about the evidence of carcinogenicity.

8 They have three categories of evidence.  
9 The first evidence is, we believe there is sufficient  
10 evidence to establish causation and they have reached  
11 that in about 30 cases. Some chemicals you may know,  
12 benzene, arsenic, cigarette smoking, DES. As I said,  
13 about 30 kinds of chemicals or, in some cases,  
14 occupational settings.

15 They then have limited evidence, a  
16 limited evidence category which says it does not rise  
17 to causation but there is some suggestions of an effect  
18 and further studies ought to be done, and then they  
19 have inadequate to classify, that's the third IARC  
20 category which says the evidence doesn't even suggest  
21 even the limited category.

22 Okay. So the quote from the MOE is,  
23 "Using IRAC terminology..." they chose  
24 to use that same terminology,  
25 "...it may be concluded that there is

1                    limited evidence of carcinogenicity in  
2                    man from exposure to phenoxy herbicides.  
3                    In terms of exposure to 2,4-D  
4                    specifically, the evidence must be  
5                    regarded as inadequate to classify it as  
6                    a carcinogen."

7                    If you read the report carefully, the  
8                    limited evidence they refer to primarily concerns  
9                    non-Hodgkin's lymphoma, although only one of these  
10                  three. The soft tissue sarcomas have been looked for  
11                  in several additional studies and seen one other time,  
12                  but in most cases you never see them again. So you  
13                  don't have any consistency.

14                  Hodgkin's disease has not been seen with  
15                  any consistency whatsoever. There is some pattern of  
16                  consistency for non-Hodgkin's lymphoma, this particular  
17                  case, but it seems the best you can say is phenoxy  
18                  herbicides might be involved there, not any specific  
19                  herbicide, and it's suggestive only.

20                  The Harvard panel of experts reaches  
21                  close to that same conclusion. There is one subtle  
22                  difference and I need to point that out. The Harvard  
23                  panel of experts' conclusion was, using case control  
24                  and cohort studies, those are two kinds of epidemiology  
25                  studies:

1                   "Epidemiologists have examined whether  
2                   human exposure to phenoxy herbicides is  
3                   associated with various forms of cancer.  
4                   While a cause/effect relationship is far  
5                   from being established, the epidemiology  
6                   evidence for association between use of  
7                   2,4-D and non-Hodgkin's lymphoma is  
8                   suggestive and requires further  
9                   investigation."

10                  Just my own editorial comment on that,  
11                  this report emphasizes throughout the problem in all of  
12                  these studies of separating 2,4-D from all the other  
13                  phenoxy herbicides. As you read the report, you find  
14                  them trying to deal with that problem. I just found it  
15                  a little surprising in the conclusion that they then  
16                  linked a suggestive evidence 2,4-D with non-Hodgkin's  
17                  lymphoma.

18                  The rest of report wouldn't seem to  
19                  suggest that, but that's in fact what they say in their  
20                  conclusions. They suggest there is very little  
21                  evidence of association between use of 2,4-D and soft  
22                  tissue sarcoma or Hodgkin's disease and no evidence of  
23                  association between 2,4-D and any other form of cancer.  
24                  So those two conclusions -- there is a slight  
25                  difference between the two, but they are basically

1       very, very close. So causation not established, based  
2       on human evidence, but certainly they are both  
3       suggesting additional studies.

4                                                                                  Animal evidence I will summarize quickly.  
5       They all looked at that. The Crump report also deals  
6       with the animal evidence. There are -- there were  
7       three sets of studies. This is summarized in the Crump  
8       report, published in the period from '69 up to through  
9       '74 on 2,4-D and certain esters of 2,4-D and amine.  
10      They were all negative, either in rats mice or both.

11                                                                                  I must say that those three studies were  
12       quickly passed by by the MOE Board of experts. They  
13       are fairly old studies and although they don't show an  
14       effect, you can't give them very much weight and I  
15       wouldn't either.

16                                                                                  There is a more recent study conducted at  
17       EPA's requests by something called the Industry Task  
18       Force on 2,4-D, that is a study in mice and rats of  
19       carcinogenic activity in a lifetime feeding study for  
20       2,4-D submitted to EPA. This has been reviewed and  
21       judged as providing insufficient evidence of  
22       carcinogenicity in mice and rats by the MOE panel of  
23       experts and by the Harvard panel as well.

24                                                                                  They did a very thorough evaluation in  
25       both cases. The MOE panel's evidence evaluation is

1       essentially good on that particular question. EPA had  
2       reached basically the same conclusions. There is one  
3       thing you ought to know, and I think Dr. Rachman noted  
4       it for you, the EPA is seeking additional animal  
5       studies on 2,4-D and it is one of the controversies  
6       that comes out of a technical issue having to do with  
7       these cancer tests.

8                   I said earlier that when you do a cancer  
9       study you try to give very, very high doses so you can  
10      be sure to predict risks if they are there because it  
11      is difficult to predict risk unless you give very high  
12      doses.

13                  The EPA judged that the \*\*ITF studies, at  
14      least in the mouse and probably in the rat, do not get  
15      to a high enough dose to be fully adequate to satisfy  
16      their requirement, what is called a maximum tolerated  
17      dose. The idea is you have got to pump up the dose  
18      until you get as high as you can so you can see the --  
19      but without threatening the lives of the animal in any  
20      other way except for the development of the cancer. So  
21      you try and increase the sensitivity.

22                  EPA does not find them acceptable, the  
23      MOE panel of experts found the rat study acceptable and  
24      it is a very, very close call.

25                  I have not gone through the studies in

1 all detail myself, so I do not have an independent  
2 opinion on the -- the MOE panel thought it was achieved  
3 in the rat but they were not sure it would have been  
4 achieved in the mouse. They may not agree with EPA  
5 when they evaluated the study, but that's all the  
6 evidence we have and that's the conclusions reached by  
7 those panels.

8 Finally I will go to a little bit of the  
9 other relevant data. This was reviewed extensively by  
10 the MOE panel of experts and less by the Harvard panel.  
11 They didn't give it as much attention as the MOE panel  
12 did. They have a long discussion of the so-called  
13 potential for 2,4-D to cause damage to genes. These  
14 are studies -- these are not cancer studies, but other  
15 kinds of studies, some of them in test tubes with  
16 bacteria, others in whole animals, and their  
17 conclusion, the MOE conclusion -- there are probably, I  
18 didn't count them, but there are probably two dozen  
19 studies on 2,4-D in the scientific literature.

20 They decided it was not genotoxic, so  
21 then finally putting all that together, both panels  
22 agree on the total weight of the evidence from the  
23 animals studies, the human studies and the general  
24 toxicity studies. The MOE panel of experts said:

25 "There is insufficient evidence to

1 support a finding that 2,4-D is a  
2 carcinogen and, consequently, there is  
3 insufficient evidence to conclude that  
4 existing uses of 2,4-D in Ontario pose a  
5 significant human health risk."

6 I will mention in a moment, as I discuss  
7 Crump now as my final discussion, that the MOE panel of  
8 experts did go a bit beyond the weight of the evidence  
9 evaluation of carcinogenicity and did some more  
10 extensive evaluations of potential risk, and I will  
11 refer to those as I proceed.

12 The Harvard panel of experts, I gave you  
13 their conclusion on cause/effect relationships, it's  
14 far from being established. And, again, they do  
15 suggest additional studies because of the possible  
16 suggestion of an association with non-Hodgkin's  
17 lymphoma that seems to appear in several studies; not  
18 all studies, but several studies.

19 Q. The cause and effect relationship  
20 they are referring to is between 2,4-D and cancer?

21 A. Yes, this is the whole -- that's what  
22 these both refer to, a question of whether the hazard  
23 evaluation -- whether 2,4-D is a potential human  
24 carcinogen.

25 Q. Now, we are referring to page 9.

1                   A. The last part of the presentation  
2       deals with Dr. Crump's report, Dr. Crump and his  
3       associates, Exhibit...

4                   Q. 716.

5                   A. 716. It is entitled Worst-Case  
6       Analysis Study on Forest Plantation Herbicide Use. It  
7       was prepared for the Department of Natural Resources of  
8       the State of Washington. They're specific to  
9       herbicides and at least three -- I have the seven that  
10      they examined in detail and three, maybe four are  
11      relevant here in Ontario, 2,4-D, glyphosate, and  
12      picloram I understand are relevant here. I don't know  
13      whether triclopyr is, I heard it might be, I don't  
14      know. The others I guess are not, but these are the  
15      ones that were of issue in the State of Washington.  
16      I must say that they looked only at aerial application,  
17      and I might emphasize that.

18                  We went through the Crump analysis, it is  
19      quite extensive, to determine whether it was conducted  
20      according to standards that we believe are appropriate  
21      for risk evaluation, the steps or the process I  
22      referred to earlier, whether they were clear in the  
23      assumption where they had to use assumptions about --  
24      in certain cases, whether they stated what they were  
25      and their basis and whether -- when they said they were

1       doing a worst-case analysis, whether they documented  
2       that in fact that was the case. So that was our  
3       analysis.

4                   They evaluated a hazard identification  
5       phase, all of the non-toxic effects of the herbicides.  
6       There are extensive toxicity reviews throughout their  
7       report. They did not discount any reported effect as  
8       irrelevant to humans that we could see, and I think  
9       there -- if anything, excessively concerned here.  
10      There are some instances where the authors of some of  
11     the reports that they reviewed concluded that a  
12     particular effect was not related to the herbicide, but  
13     it appeared and the authors may have tried to explain  
14     it away as spurious, but in the Crump report you will  
15     find consistently that they don't discount those  
16     information. So they took a very conservative view to  
17     the health evaluation.

18                  They also did something that may sound a  
19     little unusual, but it is not uncommon in risk  
20     assessment, this third bullet, they assumed, even  
21     without evidence, that all the herbicides could be  
22     potential human carcinogens, even when the evidence was  
23     negative or otherwise inconclusive. Now, why would you  
24     do that.

25                  Well, when you have a so-called negative

1       animal study, let's say, as you saw from my earlier  
2       chart, I hope, that those studies are capable of  
3       detecting fairly high risks at fairly high doses, but  
4       it is just possible that there may be a cancer risk and  
5       you couldn't detect it. So it is not uncommon in risk  
6       assessment to say: Well, what if we just missed  
7       detecting the cancer risk here, how bad -- a cancer  
8       hazard, I should say, let me be careful, what if we  
9       just missed it in this negative study, that there  
10      really was an effect, but it was just below our  
11      detection level.

12                   Well, we can answer the 'what if'  
13       question by doing a kind of risk assessment which says:  
14       I am going to assume there was an effect right at the  
15       detection limit of this experiment, and then proceed.  
16       I will show you a little bit on the dose response how  
17       that's done.

18                   I might add a couple of points here. The  
19       Ministry of the Environment panel had this -- I'm  
20       sorry, the ITF animal study that I listed earlier, as  
21       done by this Industry Task and submitted to EPA, was  
22       not available to Crump when they did their analysis.

23                   It was available to the MOE panel and  
24       they reviewed it and did with it the same sort of thing  
25       that Crump had done with the earlier cancer data; that

1       is, they assumed in their evaluation, they being the  
2       MOE panel, that there might be a risk in that study but  
3       we just missed it, and they proceeded to do a risk  
4       assessment on that basis.

5                     On chart No. 11, the dose response  
6       evaluation, in Crump they consistently use, as far as I  
7       could tell in going through all of the data, we did not  
8       review obviously all the underlying data because they  
9       cite hundreds and hundreds of studies, but where they  
10      were cited and laid out in their analysis they use  
11      always the most sensitive indicator of toxicity for  
12      non-cancer effects to establish the no-effect level, as  
13      it is called, the basis for deriving the margin of  
14      safety.

15                   They used the linear no threshold model  
16      for carcinogens that I mentioned earlier and even went  
17      a little bit beyond the linear model, they used what is  
18      called an upper statistical confidence limit on it to  
19      put more conservatism into the process. As I said  
20      before, for non-carcinogens they assumed a risk.

21                   Can I just go back to my graph for one  
22      second to give you an idea of how that can be done,  
23      2(a). For all of those herbicides where there was  
24      animal data, cancer data that were negative or of  
25      borderline significance, what Crump did was to say you

1 can calculate where those experiments run out of  
2 detection power, roughly around this risk range. So  
3 they said, what if there was an excess risk in the  
4 animal study right here, we just missed it, and that's  
5 possible. So let's assume that risk exists, then you  
6 can do the same kind of extrapolation.

7 That's what they did, and I will add to  
8 that, that the MOE panel did the very same thing except  
9 with this newer set of animal data that was not  
10 available to Crump. So both of them looked at that  
11 question.

12 I have to emphasize, 2,4-D -- I'm sorry,  
13 the MOE panel looked only at 2,4-D, Crump looke at all  
14 the other herbicides. So that's what I mean here in  
15 chart No. 11 that I was on, third bullet. For those  
16 that are not yet shown to be carcinogens but were  
17 assumed to be, they used the highest possible risk -  
18 and I put that in quotes - that you could derive from  
19 the negative data. That has to be kept in mind when  
20 you look at the risk results.

21 On the human exposure evaluation, this is  
22 probably the most complex part of the Crump report,  
23 they considered both workers, those involved in the  
24 aerial application, the mixers, the loaders, the  
25 pilots, the mechanics, the Department of Natural

1       Resources' personnel who would be nearby and what in  
2       the typical risk assessment setting is called the  
3       general population. That's the term we use for people  
4       other than those involved occupationally, bystanders I  
5       guess has been the term used here. It means the same  
6       thing, but members of the general population which  
7       would include children and older people, et cetera.

8                   They considered both of those categories,  
9       they looked at every pathway of exposure that could  
10      exist. They might have missed some, but I would be  
11      hardpressed to point one out. They looked at  
12      inhalation of the herbicides from the air, direct skin  
13      contact for workers, that's obvious, they also  
14      considered direct skin contact of bystanders who might  
15      pass through a sprayed area and into contact, say, with  
16      plants.

17                  They considered ingestion of foods, and I  
18       guess I should add here drinking water. They assumed  
19       people would drink from streams that might have been  
20       affected. Fish, wild game and berries were all  
21       considered. They presented both what they call  
22       reasonable and worst-case exposures and by that, in  
23       going through the analysis where they had data or had  
24       to use assumptions, they tried to set out very clearly  
25       what they meant by a more typical kind of exposure

1 situation, both for the worker and for members of the  
2 general population, and they also considered what might  
3 be a worst-case.

4 Let me give you one idea of what they  
5 meant by worst-case. I'm not even sure I would go  
6 quite this far in the worst-case, but here is the  
7 general population exposure that they considered, that  
8 an individual would have all of these characteristics,  
9 not just one of them or two of them, but all of them,  
10 that the individual lives continuously outdoors 24  
11 hours a days, that the pesticide -- the only pesticide  
12 that individual has contact with is that which came  
13 down at the maximum approved usage rate, not other  
14 rates, that the total body skin area would be exposed.  
15 That's an important determinator of how much gets into  
16 the body, how much of the skin area is exposed. They  
17 used the assumption of total body area. For their  
18 reasonable case, they use half the body area. That's  
19 the sort of difference they make.

20 Now, that is an assumption and we don't  
21 really know what sort of average body area is exposed,  
22 but those were their assumptions. They are probably  
23 both quite high. Well, obviously I guess the worst  
24 care is fairly high.

25 They used their data on how much of the

1       herbicides go through the skin when they are applied to  
2       the skin. There are humans studies on this question,  
3       there are animal studies on this question. They give a  
4       range of outcomes for the different herbicides and in  
5       their worst-case they assumed the high end of every  
6       reported value for the amount that goes through the  
7       skin.

8                   They had -- this person would be eating  
9       at two levels of food consumption rates, half kilogram  
10      gram per day, for example, of wild game, half kilo, was  
11      their estimate of what might be the worst-case and they  
12      used a rate that approximates more normal meat  
13      consumption based on other data for the more reasonable  
14      case. They then took where the more data that shows  
15      that the herbicides persist in different environments  
16      for differents period of time. They used for the  
17      worst-case the data shown with the longest persistence  
18      after a spraying episode and in the reasonable case  
19      they used a more average level.

20                  So those are some of the kinds of  
21      characteristics that are worked into this analysis and  
22      they are all laid out. It is quite an effort to  
23      understand that and go through the report, but it is  
24      all in there. So, again, those have to be kept in mind  
25      when doing this analysis.

1 Now, the last chart here --

2 MADAM CHAIR: Excuse me. Sorry, Dr.

3 Rodricks.

4 DR. RODRICKS: Sorry.

5 MADAM CHAIR: We normally break for lunch  
6 at twelve. Is this a good time for you to break or is  
7 it...

8 DR. RODRICKS: I will finish this in five  
9 minutes I should think, not much longer, unless you  
10 have questions. This chart, which is a summary -- it's  
11 up to you, five, ten minutes maximum.

12 MADAM CHAIR: I see we will then get into  
13 the conclusions on the analysis. I think I would  
14 rather hear the last part together.

15 DR. RODRICKS: Okay, that's fine.

16 MADAM CHAIR: Thank you very much. We  
17 will be back at 1:30. Thank you.

18 MR. CASTRILLI: Madam Chair, I am  
19 wondering if I could simply advise the parties and  
20 yourselves of the exhibits I expect I will want this  
21 afternoon or be referring to this afternoon and  
22 probably tomorrow so that everyone involved can acquire  
23 them from their offices, if necessary.

24 Exhibits 714, 715, 716, 717, 754, 789,  
25 1233, 1236, and 1237.

1                   MR. CASSIDY: Are there any transcript  
2                   volumes, Mr. Castrilli, that you intend to refer to?

3                   MR. CASTRILLI: There actually is one. I  
4                   actually may not have to refer to it, but it's the one  
5                   that's referred to, I believe it is 122.

6                   Actually, I'm sorry, I can't confirm that  
7                   at the moment, but I think it is 122.

8                   MR. CASSIDY: Are there any other volumes  
9                   or just that one as a possibility?

10                  MR. CASTRILLI: I think it is that one.

11                  MR. CASSIDY: All right. Thank you.

12                  ---Luncheon recess taken at 12:00 p.m.

13                  ---On resuming at 1:30 p.m.

14                  MADAM CHAIR: Please be seated.

15                  Mr. Cassidy?

16                  MR. CASSIDY: Good afternoon, Madam  
17                  Chair, Mr. Martel.

18                  We are prepared to continue with some  
19                  final comments from Dr. Rodricks. I anticipate we will  
20                  be approximately another 10 minutes and then Mr.  
21                  Castrilli has indicated he is prepared to proceed.

22                  Dr. Rodricks?

23                  DR. RODRICKS: I was just about to  
24                  present a small piece of the risk characterization  
25                  section of the Crump report. This table deals with the

1 highest risk that they found. This was individuals  
2 involved in mixing and loading pesticides for forest  
3 application and this table -- this chart; that is,  
4 overhead No. 13, pertains to use of that group.

5 Another very, very important piece of  
6 this that must be specified, this is the way Crump  
7 presented the data in their summary. This risk  
8 pertains to the risk associated with a single spray  
9 application. Now, obviously that wouldn't be the total  
10 risk, you have to go back into the report to find the  
11 total risk and the total risk would be -- I will also  
12 report that to you.

13 Because we are dealing with risks which  
14 are basically directly proportional to exposure, you  
15 can take the risk for a single spray application and  
16 answer the question for any other number of  
17 applications you might want to look at for, let's say,  
18 workers who are involved in spraying and they did that  
19 in their report and I will...

20 Anyway, for the single spray application,  
21 for 2,4-D, the worst-case risk of cancer that they  
22 found - this is what we call assumed because we are  
23 assuming that 2,4-D was a carcinogen - was of the order  
24 of - I would say less than, because remember also the  
25 methodology places an upper bound level on the risk -

1 one in 25 million over a lifetime.

2                   There are many different ways that one  
3 can look at the question of total risk. In Crump's  
4 report they relied upon data from the Department of  
5 Natural Resources in the State of Washington on the  
6 periods of time people in the pesticide application  
7 business hold jobs, how many spray events they might be  
8 involved in over a lifetime. In the Crump report,  
9 there is a table at the rear of the report that deals  
10 with this.

11                  I may want to just mention that number,  
12 may I? I guess I should have written it down, sorry.  
13 I can find it quickly. Table 11-15 of the Crump report  
14 contains one estimate of total risks that might be --  
15 cancer risks that might be associated with various  
16 herbicides for different occupational categories, the  
17 pilot, the loader, the mixer loader, the mechanic, the  
18 observer.

19                  For the worst-case exposure, those risks  
20 rise to about one in 100,000 over a lifetime for the  
21 loader, for the mechanic and for observers they are  
22 still in the order of one to three per million over a  
23 lifetime. Now, that would be more specific to the  
24 State of Washington.

25                  I might add, then, that on this

1       particular issue also, 2,4-D, as I mentioned before,  
2       the Ministry of the Environment's expert panel also  
3       made a similar kind of analysis for 2,4-D and for  
4       workers. Results of that analysis are presented on  
5       page 51, Table 8 of the MOE report.

6                     This has some advantages over the Crump  
7       analysis because, No. 1, it relies upon the more recent  
8       animal cancer bioassay data that I mentioned before for  
9       their worst-case analysis. Remember, this was a  
10      negative study or not sufficient evidence of  
11      carcinogenicity, as the panel named it, but they went  
12      and performed this analysis on the assumption that it  
13      might be a carcinogen.

14                   They also have had available, and it is  
15      summarized in the MOE report, some more recent data and  
16      it is Canada specific -- Ontario specific on  
17      occupational exposures to 2,4-D incurred during the  
18      application of either aerial application or exposures  
19      that are incurred from individuals who are applying  
20      2,4-D from backpack, hand-held applications.

21                   Their analysis -- and they also looked at  
22      the question of the number of days over a lifetime when  
23      individuals might be involved in these spray operations  
24      in Ontario. They had that information and they had a  
25      citation to that. I can only -- I can't verify it is

1       correct, but it is a citation to a department of -- may  
2       I just check.

3                   Yes, I'm sorry. On page 20 of the MOE  
4       report they refer to a survey from DHS, 1987. It was  
5       the basis for the number of days in a lifetime that  
6       exposures would occur to workers involved in the  
7       application of 2,4-D in Canada. So there was a  
8       Canadian -- Ontario specific survey.

9                   Their risk numbers are in the same range  
10      as Crump's. The risk numbers for a full lifetime risk,  
11      the highest risk they found was for the backpack  
12      sprayer. Those risks are reported in Table 8 of the  
13      MOE report, in the order of five to eight per million  
14      over a lifetime. Those are the highest risks they  
15      found in an occupational setting, and for those  
16      involved in aerial application, the mixer, loader  
17      risks, as in Crump, were the highest, but in the MOE  
18      analysis they were a little under one in a million over  
19      a lifetime; somewhat less than Crump reports, but  
20      clearly in about the same range. So we have those two  
21      analysis which are in fairly close agreement.

22                  Just for perspective, in the United  
23      States at least, and this comes out in the MOE report  
24      for occupational carcinogens, occupational carcinogens  
25      in the United States are regulated by the Occupational

1 Safety and Health Administration, and part of the basis  
2 for regulation of such carcinogens is a risk assessment  
3 of the same type I have talked about here.

4 For workplace exposures, the OSHA, as we  
5 call them, have made decisions on about a dozen  
6 carcinogens over the last 10 years have not sought to  
7 reduce occupational -- lifetime occupational risks for  
8 carcinogens below about one per thousand. That's about  
9 as low as they get, one per thousand.

10 The MOE report presents a couple of  
11 examples, but there are many more to choose from. Now,  
12 that is a policy decision to be sure, but these risks  
13 are certainly well below - 'these', that is pertaining  
14 to 2,4-D under both analysis - are well below what at  
15 least has been accepted by OSHA for occupational  
16 carcinogens. That may not be the only standard, but  
17 that's certainly one.

18 The Crump report deals further with  
19 glyphosate and picloram, a similar analysis, yields  
20 risks again from a single application very, very much  
21 less than one in 25 million for both of these combined.  
22 These are lowest risk figures in this chart and, again,  
23 if you refer to Table 11-15 of the Crump report where  
24 they deal with total lifetime risks in Washington,  
25 these rise; that is, glyphosate and picloram, rise to a

1 maximum of about one to five per hundred million over a  
2 lifetime. They are extremely small risks.

3 They also deal with margins of safety for  
4 reproductive and teratogenic effects and, again,  
5 remember this is the worst-case analysis for  
6 occupational exposures. I should add that there is  
7 also a great deal of information about the general  
8 population and all the risks are much lower than these.

9 Margins of safety for reproductive and  
10 teratogenic effects, this is the difference between the  
11 no-effect level, the maximum dose in which you see no  
12 toxicity, and the actual exposure to 2,4-D. They  
13 reviewed all the reproductive and teratogenic toxicity  
14 data for these three and the other herbicides and found  
15 margins of more than 100 for 2,4-D, more than 400 for  
16 glyphosate and more than 20,000 for picloram. These  
17 vary because these materials have different degrees of  
18 toxicity.

19 For other toxic effects, they also review  
20 just sort of general toxicity of these materials other  
21 than reproductive or teratogenic effects. They found  
22 margins of safety from more than five 2,4-D for the  
23 worst-case worker, more than 100 for glyphosate and  
24 more than 2,000 for picloram.

25 I need to comment on the five a little

1       bit. This is a worker population and a worst-case  
2       analysis. Crump characterizes this very, very  
3       carefully because they did something a little unusual  
4       here. They chose for the no-effect level for -- what I  
5       am just calling other toxic effects, it turns out in  
6       the case of 2,4-D it will effect the kidney adversely  
7       at a sufficiently high dose, so it's just kidney  
8       toxicity.

9                  They took data from what is called a  
10       90-day study where the animals are exposed continuously  
11       over 90 days, that is called a subchronic study, to  
12       derive a no-effect level for this material and took the  
13       margin of safety from that, but now recall we are  
14       dealing now with exposures which are highly  
15       intermittent and in the human lifetime comprising a  
16       much smaller fraction over the lifetime. So there is  
17       some additional margin of safety in there beyond the  
18       five. It is not really possible to estimate that.

19                  I might also add, and this comes out of  
20       the MOE report because the no-effect level that  
21       Crump -- the toxic effect level that Crump referred to  
22       for systemic effects for 2,4-D; that is, for these  
23       other toxic effects, came from this 90-day study I  
24       referred to. That 90-day study was a study submitted  
25       to the Environmental Protection Agency by this Industry

1 Task Force on 2,4-D as a study that was to be used to  
2 make decisions about how to design the long-term cancer  
3 study they were to do. It is called a screening study,  
4 where you try to figure out from results of that study  
5 how to dose the animals for a lifetime.

6                   What the MOE report points out is that  
7 when -- and that was not available to Crump, when that  
8 long term, two year exposure study actually took place,  
9 there was no effect on the kidney seen at the dose  
10 where you saw something in the 90-day study. No effect  
11 whatsoever.

12                  The MOE doesn't try to explain that,  
13 nobody has tried to explain that very much except so  
14 say that - and this is in the MOE report - that in all  
15 studies there are effects you will see in various  
16 organs that occur spontaneously in animals and you try  
17 to separate those from effects due to the chemical. In  
18 this case, we are not sure why we had the observation  
19 in 90 days of something going on in the kidney and no  
20 such effect over -- we the exposure was longer at the  
21 same dose. So there is a little uncertainty there  
22 about where that no-effect level is, but the long term  
23 study should give us some comfort.

24                  The reasonable exposure scenarios, the  
25 more typical exposures, of course the risks are

1 smaller, roughly on average 80 times smaller than the  
2 worst-case cancer risks and safety margins are about  
3 four times larger for the reasonable exposure  
4 situation. Again, these are for workers, the general  
5 population much less.

6 I will close then just to mention some  
7 other characteristics of the Crump analysis. We were  
8 asked to comment on the general procedures, the quality  
9 of the report and the group. Dr. Crump is one of  
10 the -- as I say, is an internationally recognized  
11 expert in chemical risk assessment. That is kind of an  
12 understatement.

13 He is one of the people whose  
14 publications during the late 60s and early 70s really  
15 gave rise to much of what we do in risk assessment.  
16 So he is really quite widely acknowledged as an expert  
17 in the area.

18 The methodology used conforms in all  
19 respects to that recognized, at least in the United  
20 States, as an appropriate way to evaluate risks.

21 Q. I understand that you wish to file  
22 copies of the guidelines that are used in the United  
23 States for assessing carcinogen risk?

24 A. Yes. The Environmental Protection  
25 Agency has actually published from time to time reviews

1 or what they call guidelines on how they go about the  
2 cancer risk assessment process.

3 MR. CASSIDY: I have a copy of those to  
4 provide to the Board, Madam Chair, as the next exhibit  
5 which would be Exhibit 1246, entitled Guidelines for  
6 Carcinogen Risk Assessment, dated September 24, 1986  
7 published by the U.S. EPA.

8 ---EXHIBIT NO. 1246: Document entitled Guidelines for  
9 Carcinogen Risk Assessment, dated  
September 24, 1986 published by  
the U.S. EPA.

10

11 DR. RODRICKS: With respect to the  
12 worst-case analysis, bullet three on this last chart,  
13 we couldn't find anything in their analysis that I  
14 wouldn't consider worst-case for their worst-case  
15 scenario. It seemed to be appropriately conservative,  
16 in some cases more conservative than I would have  
17 carried out. More conservative in procedures that were  
18 adopted than I would have adopted.

19 There is no clear single definition of  
20 what you mean by a worst-case analysis and how you put  
21 together the data to reach a conclusion about  
22 worst-case. I showed you some examples from the  
23 report. They seemed to be a bit extreme; but given the  
24 outcome of the assessment, I don't think that's  
25 problematic, but this seems to be -- excessively

1       pessimistic, as I've said here.

2                          One of the reasons you do a worst-case  
3       analysis and risk assessment is that it is generally  
4       easier to do, requires less data and information  
5       because you could substitute some pretty extreme  
6       assumptions like the hundred per cent body exposed sort  
7       of assumption, which I guess would have to be read as  
8       as a worst-case assumption. You can do that sort of  
9       thing in a worst-case analysis without collecting for  
10      some kinds of information a lot of data. So this is  
11      not an uncommon practice.

12                       If the result of that very worst-case  
13      analysis yields risks which are extremely low, that's  
14      important information because it says -- gives you a  
15      couple of clues, the most important one of which it  
16      tells you whether it is really worthwhile proceeding to  
17      get additional information. If the risks under a sort  
18      of very worst-case assumption seemed to be very, very  
19      low, then that raises a question whether it is really  
20      valuable to learn something new. So that's one of the  
21      kinds of uses.

22                       The only qualification on its  
23      comprehensiveness, I think it's reliable, is that it  
24      was done in -- completed in '86 and would not have  
25      covered any literature like the ITF study that I

1 mentioned since the time it was done, but in other  
2 respects it seems quite comprehensive to us.

3 I think that's all I have.

4 MR. CASSIDY: That completes the evidence  
5 of this panel in-chief, Madam Chair.

6 MADAM CHAIR: Thank you, Mr. Cassidy.

7 Mr. Castrilli?

8 DR. RACHMAN: Madam Chair, while we are  
9 making this change can I be excused for a moment.

10 MADAM CHAIR: Certainly.

11 MR. CASTRILLI: Madam Chair, you can tell  
12 someone much taller than I was at the podium a moment  
13 ago. I will just be one more minute.

14 CROSS-EXAMINATION BY MR. CASTRILLI:

15 MR. CASTRILLI: Q. Dr. Rachman, can we  
16 begin with you at page 14 of your evidence. You are  
17 discussing there the special review process under  
18 FIFRA. I just want to clarify one comparatively minor  
19 point.

20 In your summary on those pages with  
21 respect to the special review process, my understanding  
22 is that that process includes a consideration of  
23 whether a pesticide that is already registered on the  
24 basis that it will not cause unreasonable adverse  
25 effects on human health may in fact be causing such

1 effects?

2 DR. RACHMAN: A. That's correct. If I  
3 understand your question correctly, a pesticide that  
4 has already been registered and, therefore, which has  
5 been found to satisfy the no adverse effect criterion,  
6 may subsequently go into special review.

7 That scenario would apply if additional  
8 information were involved that changed the scientific  
9 picture.

10 Q. Fine, thank you for that  
11 clarification. And I understood your testimony on this  
12 issue as well, that the result -- or the results of a  
13 special review could include, for example, continued  
14 unmodified registration, that's one option?

15 A. That is an option, yes.

16 Q. And a further possible option might  
17 be use restrictions or label modifications?

18 A. Yes. The point I was trying to make  
19 is that the agency might take a variety of actions.  
20 They would choose the one that would be most  
21 appropriate to mitigate the risk that had been  
22 identified as significant in the special review.

23 Q. And a further result of the special  
24 review process might be cancellation or suspension of  
25 the registration; is that correct?

1                   A. It could be a proposal from the  
2 agency for cancellation or suspension and then, of  
3 course, those procedures would have to be played out  
4 under the provisions of the law and the regulations.

5                   Q. Okay, thank you. Now, I just wanted  
6 to refer you as well, Dr. Rachman, to page 20 of your  
7 evidence. Now, I understand that -- I'm sorry, Dr.  
8 Rachman, we are in particular referring to your  
9 discussion on that page regarding the special review  
10 process as it applies to 2,4-D, so we are looking at  
11 the bottom of that page.

12                  As I understood your testimony this  
13 morning, the information contained on page 20 and over  
14 .to page 22 really now has to be considered in light of  
15 what is now Exhibit 1242, which is the--

16                  A. The latest, yes.

17                  Q. --Federal Register for October 13,  
18 1989; is that right?

19                  A. That's correct.

20                  Q. And as I understand Exhibit 1242,  
21 what the agency is saying is that it is postponing its  
22 decision not to initiate a special review of 2,4-D; is  
23 that correct?

24                  A. That's my understand of that, yes.

25                  Q. And as I understand it, among the

1       reasons given by the agency for that decision to  
2       postpone a decision not to initiate a special review  
3       include a number of ongoing epidemiological studies; is  
4       that correct?

5                     A. Yes, that's my understanding.

6                     Q. And that's reflected, for example, in  
7       the summary that you, I believe, read into the record  
8       at page 42,032 of Exhibit 1242? It is the left-hand  
9       column of that exhibit. Do you not have a copy of the  
10      exhibit?

11                  A. I am getting my numbers confused  
12      here.

13                  Q. All right.

14                  A. Just one moment. We have a copy of  
15      that exhibit here.

16                  MADAM CHAIR: Do you want to repeat that,  
17      Mr. Castrilli?

18                  MR. CASTRILLI: Madam Chair, we are  
19      referring to Exhibit 1242 which the U.S. Federal  
20      Register -- or I should say U.S. EPA Federal Register  
21      document dated October 13, 1989 and we are referring to  
22      the summary paragraph which is called Summary.

23                  MADAM CHAIR: Thank you.

24                  MR. CASTRILLI: The first column on page  
25      43,032.

1 DR. RACHMAN: Okay. The summary  
2 paragraph being the first full paragraph?

3 MR. CASTRILLI: Q. Yes. Actually, it is  
4 the paragraph called Summary as it happens.

5 DR. RACHMAN: A. Mr. Castrilli, I'm  
6 terribly sorry, could you please repeat this question?  
7 I have just gotten lost.

8 Q. Yes. As I understand it, the  
9 reason -- among the reasons the agency has given for a  
10 decision to postpone its decision not to initiate a  
11 special review of 2,4-D includes the fact that there  
12 are a number of epidemiological studies which are  
13 ongoing and which the agency wishes to consider before  
14 it makes a final determination; is that correct?

15 A. Yes.

16 Q. Thank you.

17 MADAM CHAIR: Excuse me, Dr. Rachman.  
18 Are they only the two studies that we heard about in  
19 Nebraska and in Iowa?

20 DR. RACHMAN: Those are the two studies  
21 that are specifically mentioned in this notice, Madam  
22 Chair.

23 If you will look at page 42,034, the  
24 left-hand column, the top paragraph says Forthcoming  
25 Data, and they describe the studies in that paragraph.

1       One from eastern Nebraska, the other from Iowa and  
2       Minnesota, both NCI studies in progress.

3                   MADAM CHAIR: And then the final sentence  
4       says:

5                   "Other epidemiological studies which may  
6       provide some information about 2,4-D are  
7       being performed or planned by NCI."

8                   DR. RACHMAN: Yes. I have no information  
9       about what those studies might be.

10                  MR. CASTRILLI: Q. And just bear with me  
11       for one moment, Dr. Rachman. The current status,  
12       therefore, of 2,4-D in the U.S. EPA regulatory process  
13       is that no final decision has been made on whether to  
14       initiate a special review; is that correct?

15                  DR. RACHMAN: A. Yes. I would agree  
16       with that. Whether that constitutes regulatory status,  
17       I think it's a legal question which I really can't  
18       answer. I mean, no decision has been made as of this  
19       time that a special review is warranted and it's going  
20       to stay that way until this additional information is  
21       reviewed.

22                  Q. Dr. Rachman, we are still at page --  
23       actually, let's go to your summary or your -- what do  
24       you call it, your executive summary?

25                  This would be (iv), paragraph 12.

1                           MR. CASTRILLI: Madam Chair, in case that  
2 was not clear, it's page (iv), paragraph 12, a portion  
3 of the executive summary for this exhibit which is  
4 Exhibit 1239.

5                           Q. Dr. Rachman, let me just read the  
6 paragraph into the record that I am referring to.

7                           "The EPA has not identified any risks  
8 with respect to forestry use in the  
9 United States of the chemical pesticides  
10 approved for forestry in Ontario and has  
11 not imposed, with respect to these  
12 pesticides, any form of risk mitigation  
13 requirement related to human health  
14 effects from forestry uses."

15                          Dr. Rachman, I gather, therefore, by  
16 deduction that that is the situation, in your opinion,  
17 with respect to 2,4-D as well?

18                          DR. RACHMAN: A. Yes, that's correct.  
19 My understanding is that no risks have been identified  
20 from the forestry uses of 2,4-D and no risk mitigation  
21 measures have, therefore, been imposed.

22                          Q. Dr. Rachman, can you confirm for me  
23 that the U.S. EPA is not the only federal agency in the  
24 United States that make decisions with respect to the  
25 health and environmental risk posed by herbicides such

1 as 2,4-D?

2 A. No, in fact I cannot confirm that,  
3 Mr. Castrilli. The EPA has the responsibility for the  
4 federal registration of the pesticide and that  
5 registration involves the determination of whether or  
6 not unreasonable adverse effects are likely to occur  
7 under the proposed conditions of use, as I explained in  
8 my testimony.

9 Now, there are other agencies, both  
10 federal and state and even local, that make decisions  
11 in the United States with respect to the use of various  
12 pesticides and the conditions of use of various  
13 pesticides. They may do independent reviews of data,  
14 but their decisions do not affect the federal  
15 registration status in any way.

16 Q. Let me be more specific. Are there  
17 other federal agencies in the United States that make  
18 decisions with respect to environmental health risks,  
19 if I can use that term, posed by herbicides such as  
20 forestry -- excuse me, such as 2,4-D for forestry use?

21 A. The United States Forest Service,  
22 which is part of the U.S. Department of Agriculture,  
23 does environmental impact analyses of the proposed  
24 forestry uses in the United States.

25 As I'm not an expert in this area, my

1 understanding is that under the requirements of the  
2 National Environmental Policy Act major undertakings  
3 that are proposed by government agencies -- the  
4 environmental impacts of those proposed undertakings  
5 have to be evaluated, very similar to the proceeding  
6 that we are taking part in here.

7 In that sort of proceeding, the forest  
8 service will review and evaluate evidence relating to  
9 environmental health risk of pesticides. I will just  
10 leave it at that.

11 Q. Thank you. Do you have Exhibit 1237  
12 before you? That would be the Ozark -- it is the very  
13 last document on your desk.

14 A. The big one?

15 Q. Yes. Not quite the Toronto phone  
16 directory but I guess it would do in a pinch.

17 A. Yes.

18 Q. Dr. Rachman, are you aware that the  
19 various regions -- sorry. Let me withdraw that  
20 statement since that it is not true in the context of  
21 the question I was about to ask you.

22 Would you agree with me that there is at  
23 least one forest region in the United States U.S.  
24 Forest Service System that has made a decision about  
25 whether to permit 2,4-D use?

1                   A. I have not --

2                   Q. For forestry purposes?

3                   A. I have not had an opportunity to  
4       review this document, Mr. Castrilli, so I cannot  
5       confirm that for you.

6                   Q. Let me refer you to one or two pages  
7       very briefly from that exhibit. Looking at (ii) --  
8       sorry, the chapters are divided by large Roman  
9       numerals, so we are looking initially (II)-(LV), That  
10      would be the page number. We are looking at Item 2 on  
11      that page, which states:

12                  "Only herbicide formulations (active  
13                   and inert ingredients) and additives  
14                   registered by EPA..."

15                  Just stopping there, Dr. Rachman. Your  
16      understanding would be that that is under FIFRA; is  
17      that correct?

18                  A. That's correct.

19                  Q. And continuing in the sentence:

20                  "...and approved by the forest service  
21                   for use on national forests are applied."

22                  Just stopping there. Would you confirm  
23      that that basically describes what you have just  
24      indicated to the Board a moment ago, that certain  
25      forest services as a result of the NEPA process are in

1       a position to make decisions about what herbicides are  
2       used in certain national forests. Is that your  
3       understanding?

4                     A. Yes, that would be my understanding.  
5       I would like to point out, though, that the decisions  
6       of whether or not or when to use a particular pest  
7       management option fall in the realm of risk management  
8       that we discussed where more is taken into  
9       consideration than simply the scientific findings of  
10      risk.

11                  There are policy issues at stake here and  
12      I'm sure that part of the decision that the forest  
13      service makes in deciding to use any particular  
14      pesticide takes into account public opinion and a lot  
15      of other things.

16                  MR. MARTEL: You are saying it is not  
17      necessarily all due to the toxicity, if I can use that  
18      term, of the substance that they are banning, in other  
19      words?

20                  DR. RACHMAN: Yes.

21                  MR. MARTEL: There are other motives.

22                  DR. RACHMAN: That would be my  
23      understanding based on similar documents that I have  
24      seen in the past.

25                  MR. CASSIDY: Mr. Martel, I wonder if I

1        might ask you, when you are speaking if you could  
2        perhaps move your microphone just a little bit closer.  
3        Thank you very much.

4 MR. CASTRILLI: And if the reporter  
5 cannot hear me, please let me know.

6 Q. Dr. Rachman, can I now ask you to  
7 turn to page -- this would now be (xii) in Exhibit  
8 1237. A unique number system. Actually, it's not such  
9 a small number but it is (xii).

10 Dr. Rachman, this is under a general  
11 heading Environmental Consequences and looking at the  
12 first subheading on the page entitled health -- excuse  
13 me, entitled Human Health and safety, I will read the  
14 paragraph into the record and I would like to ask you a  
15 question about this paragraph.

16                         "All herbicides and additives  
17                         investigated provide ample margins of  
18                         safety for the public when applied using  
19                         typical rates and methods. However,  
20                         because 2,4-D, 2,4-DP..." and two other  
21                         herbicides, some of which I may not be able to  
22                         pronounce, but in any event they are not used in this  
23                         hearing.

24                         "....have lower margins..." would never  
25                         be used in this hearing and are not proposed to be used

1       in the area of the undertaking,

2                 "...have lower margins of safety or pose  
3                 possible environmental risks they were  
4                 not considered for use in the  
5                 Ozark/Ouachita Mountains area."

6                 I will just read the last sentence:

7                 "In general, worker exposure is reduced  
8                 by aerial application."

9                 Were you aware, Dr. Rachman, that this  
10          particular U.S. forest service was not -- or is not  
11          going to permit the use of 2,4-D in the Ozarks because  
12          of lower margins of safety or possible environmental  
13          risks?

14          A. No, I was not aware of that.

15          Q. Dr. Rachman, can I now refer you to  
16          Exhibit 1236. This would be a document, it look likes  
17          that. It is entitled Record of Decision, USDA Forest  
18          Service. It is dated March 5, 1990. We are looking at  
19          page 7. At page 7 we are looking at the middle of the  
20          page, the paragraph 1 that's in brackets that begins:  
21          "Only herbicides..."

22                 Do you have the paragraph?

23          A. (nodding affirmatively)

24          Q. I will read that into the record.

25          "Only herbicides with least envir --

1 excuse me.

2 "Only herbicides with least health  
3 and environmental risks may be applied  
4 and only at most lowest effective rates."

5 There is a reference to the final EIS that I referred  
6 you to a moment ago.

7 "The herbicides that may be used are..."  
8 and they are identified there. They include for the  
9 purpose of this undertaking:

10 "...herbicides such as glyphosate,  
11 hexazinone, picloram (only products  
12 formulated without 2,4-D)..."

13 It goes on to identify several other  
14 herbicides that are going to be permitted to be used in  
15 that forest region and also identifies a number of what  
16 appear to be inert ingredients that will be permitted.

17 Dr. Rachman, were you aware that in  
18 addition to not permitting 2,4-D use in its own right,  
19 that the regional forester for the U.S. Forest Service  
20 in the Ozarks has decided not to permit the use of  
21 picloram if it is formulated with 2,4-D?

22 A. No, I was not aware of that.

23 Q. Can I refer you now to your evidence  
24 again, Exhibit 1239, and we will be looking at page 53.

25 Actually, Dr. Rodricks, to be fair to

1 you, I think it actually contains a better summary, we  
2 might also look at page (viii) of your evidence,  
3 paragraph 22.

4 DR. RODRICKS: A. Paragraph 22?

5 Q. Yes, that's correct.

6 Dr. Rachman -- Dr. Rodricks, excuse me,  
7 on that page you indicate that there are several  
8 uncertainties attached to the Kansas study and its  
9 results and you summarize the uncertainties in  
10 paragraph 22 and you expand upon them in pages 50 to 56  
11 of your evidence. I believe you restate the concern  
12 with respect to uncertainties at page 53 of your  
13 evidence.

14 - Just as a general proposition, Dr.  
15 Rodricks, would it be fair to say that in general there  
16 are uncertainties associated with every epidemiology  
17 study?

18 A. I think that's probably a pretty good  
19 generalization, yes.

20 Q. And in particular with respect to the  
21 Kansas study, can you confirm for me that it was  
22 reviewed by both the EPA and outside consulting  
23 epidemiologists and was found to be well designed and  
24 well executed?

25 A. I know of no serious criticism of the

1           design or execution of that study.

2           Q. Just to -- I'm sorry, I didn't mean  
3       to cut you off.

4           A. Are you referring to specific  
5       statements about design?

6           Q. Yes. Let's look at Exhibit 1242?

7           A. 1242.

8           Q. That's the exhibit you filed this  
9       morning. We are looking at page 42,033. Dr. Rodricks,  
10      we are looking at the first column under the heading  
11      Epidemiologic Evidence and we are looking at the  
12      last -- next to last sentence on the page.

13           A. Yes, I see that.

14           Q. And the statement reads:

15           "The study..." and this is of course a  
16      reference to the Kansas study,

17           "...was reviewed by EPA and consultant  
18      epidemiologists and found to be well  
19      designed and executed."

20           Would you agree with that assessment, Dr.  
21      Rodricks?

22           A. Yes, I think -- yes, generally.

23           Q. Just looking at the last sentence on  
24      that page, let me read that that into the record.

25           "However, after an indepth evaluation,

1                   EPA concluded that the study did not  
2                   provide sufficient evidence of a link  
3                   between 2,4-D and NHL..."

4                   Dr. Rodricks, you realize that in Canada  
5                   NHL is the National Hockey League.

6                   A. In the U.S. as well.

7                   Q. That's true.

8                   "...to pursue regulatory action at that  
9                   time."

10                  Now, just stopping there. I would like  
11                  your opinion on the sentence and what its import is.

12                  Would it be -- is your interpretation of  
13                  that -- or is a fair interpretation of that sentence  
14                  that what EPA is stating is that they are acknowledging  
15                  that there are limits to what one can expect from any  
16                  single epidemiology study? Is that a fair  
17                  interpretation of that sentence?

18                  A. Well, it's a little more specific  
19                  than that. I mean, I read it to say that they have  
20                  looked at it deeply and the study by itself does not  
21                  establish a link, in this case, between 2,4-D exposure  
22                  and a risk of non-Hodgkin's lymphoma sufficient to  
23                  pursue regulatory action.

24                  So it is a little more specific, but you  
25                  have the general intent of it.

1                   Q. All right, thank you. If I might,  
2 Dr. Rodricks, I would like to put another general  
3 proposition to you. By some time tomorrow I will get  
4 down to specifics.

5                   MR. CASSIDY: You have until Friday.

6                   MR. CASTRILLI: That's my understanding,  
7 yes.

8                   Q. As a general proposition, Dr.  
9 Rodricks, would it be fair to say that evidence of  
10 carcinogenic activity of a chemical agent can be  
11 obtained from epidemiological studies when evaluation  
12 of the observation shows that the chemical agent causes  
13 an increased incidence of neoplasms?

14                  DR. RODRICKS: A. Well, the critical  
15 word there is 'causes'. If you can establish  
16 causation, as I discussed in my presentation this  
17 morning, that is not an easy thing to do with  
18 epidemiologic methodologies, but if you can establish  
19 causation and if you have reached that point through a  
20 series of studies where you see excesses of a certain,  
21 as you put it, neoplasm or cancer, then, yes, it can  
22 happen.

23                  As I said, there are approximately 30  
24 chemicals or mixtures of chemicals where such causal  
25 links have been established, not including 2,4-D.

1                   Did I get your question right?

2                   Q. I think you had the gist of the  
3                   question. Let me pursue this a moment with you for a  
4                   moment. Would it also be fair to say that evidence of  
5                   carcinogenic activity of a chemical can be obtained  
6                   from such human studies when the evaluation of the  
7                   observations also shows that the agent causes a  
8                   decrease in their latency period?

9                   A. Well, that is one piece of evidence.  
10                  I doubt if that -- if you mean from a single study an  
11                  observation of a decreased latency period associated  
12                  with an exposure, decreased time from first exposure to  
13                  the finding of the tumour, in a single study as sole  
14                  evidence of causation that would be highly unlikely,  
15                  but it is one of several things you'd look at.

16                  I didn't mention latency in my five  
17                  criteria this morning, but it is subsumed under the  
18                  dose response relationship.

19                  Q. I'm sorry, you are competing with a  
20                  fire engine. I missed the last part of that.

21                  A. I didn't specifically -- if you  
22                  remember my five criteria from the overhead this  
23                  morning for evaluating evidence of causation, one of  
24                  those was increasing risk with increasing exposure.

25                  I perhaps should have added, and it is

1       subsumed under this, that if you also see decreased  
2       latency; that is, decreased time from first exposure to  
3       the first observation of a case with increasing  
4       exposure, that is a similar piece of confirmatory  
5       evidence.

6                     But if your question was, if you saw that  
7       in a single study and that was the only evidence you  
8       had to link an exposure to a disease, that would not be  
9       sufficient by itself. It is just one of several things  
10      one would want to see.

11                  Q. I perhaps misled you as to the focus.  
12       I wasn't in that particular question focusing on one  
13       study per se. I think I used the plural, or I hope I  
14       use the plural.

15                  Your would your answer change if you now  
16       understand the question to relate to more than one  
17       study?

18                  A. Could you do the question again,  
19       please? The question was...

20                  Q. Sure. Is evidence of carcinogenic  
21       activity -- or can evidence of a carcinogenic activity  
22       of a chemical be obtained from epidemiologic studies,  
23       plural, when evaluation of the observation shows - I  
24       think I asked you initially - an increase of incidence  
25       of neoplasms; and, secondly, a decrease in their

1 latency period?

2                   A. If that were found in different  
3 studies, preferably done in different populations with  
4 different study methods consistently, that would be  
5 quite powerful evidence, yes.

6                   Q. All right, thank you. Would it be a  
7 fair statement that -- or is it a statement you can  
8 agree with, that clinical signs of cancer can be  
9 delayed for a long time after initial exposure to a  
10 carcinogen or to carcinogens?

11                  A. Yes, that is the latency period,  
12 so-called, and it can very long for carcinogens,  
13 perhaps up to 40 years in some cases from the time of  
14 initial exposure until the appearance of the disease.

15                  Q. And would it be fair to say that the  
16 latency period can be from approximately five years to  
17 40 years from initial exposure until the disease  
18 appears?

19                  A. There are some agents where a latency  
20 as short as five years has appeared. I would guess the  
21 average to be in the 20- to 25-year range and there are  
22 some that go up to 40 years. Some asbestos producing  
23 cancers, for example, go up to 40 years. So there is  
24 quite a range.

25                  Q. Would you also agree with the

1 proposition that evaluation of epidemiologic studies  
2 requires a knowledge of the smallest possible increase  
3 in tumor incidence detectable under the conditions of  
4 the study or studies?

5                   A. I think your question has to do with  
6 the power of the study to find an effect. I mentioned  
7 in my - if you remember - graph this morning where I  
8 showed that even in animal studies and also in  
9 epidemiologic studies there is just a limit to the rate  
10 of disease that can be detected. It is a function of a  
11 number of factors, most importantly the size of the  
12 population which you are able to study.

13                  So what you are asking is whether one  
14 could estimate the power of a study to detect an  
15 effect; that is, if a study is negative, what risk  
16 could it have missed, and there are -- sometimes from  
17 epidemiology studies there are data available that  
18 allow you do that, although it is not an easy thing to  
19 do when you do not have exposure information,  
20 quantitative exposure information, but I've done that  
21 sort of calculation myself for some agents, but whether  
22 you can do it depends on the kinds of information you  
23 have available from the epidemiology study. It is not  
24 always possible.

25                  Q. Okay. I gathered from your answer to

1       my question that this type of information is of  
2       critical importance in the evaluation of apparently  
3       negative studies?

4                     A. Well, you should try to do it, I  
5       agree with that, to see whether the negative study  
6       would have -- how much of an effect a negative study  
7       could possibly have missed, yes. But I also emphasize  
8       that it is not always easy to do if you don't have the  
9       data.

10                  Q. Dr. Rodricks, we are still keeping  
11       this at a comparatively general level. Would it be  
12       fair to say that substances widely distributed in  
13       commerce or the environment are difficult to study by  
14       epidemiologic methods in part because it is often  
15       impossible to identify unexposed groups as controls?

16                  A. Your premise was substances that are  
17       very widely distributed in the environment?

18                  Q. Yes.

19                  A. Well, you may be able to discover  
20       occupational exposures, this is typically the case.  
21       They are much more intense than general environmental  
22       exposures and even though there is -- even though you  
23       can't say that the exposure in the general population  
24       is zero, it still may be small relative to, let's say,  
25       an occupational setting.

1                   So there may be opportunities if you can  
2 identify the appropriate occupational group to examine.  
3 So I think it very much depends on whether you have  
4 that opportunity.

5                   Q. Let me try this one again and use a  
6 different example. This is again with respect to  
7 substances that are widely distributed in the  
8 environment. We will forget about commerce for the  
9 moment.

10                  Do I understand your testimony to be --  
11 or indicate if you can agree with me that for  
12 substances of that type it's particularly difficult to  
13 study them by epidemiologic methods or to separate --  
14 because it is impossible to separate out groups with  
15 high and low exposure?

16                  A. And my answer was that you might be  
17 able to find occupational groups, people who are, say,  
18 in the manufacturing of the material who have very much  
19 higher exposures than the general proposition  
20 population.

21                  So you still may have the opportunity to  
22 study them, but if there is no such group or you cannot  
23 distinguish groups based on significant differences in  
24 exposure, then it's probably a fairly futile effort to  
25 try to investigate that.

1                   Q. And I think you've already indicated  
2                   that -- or maybe you haven't, let me just put the  
3                   proposition to you.

4                   Is it a fair statement to say that the  
5                   problem of adequate controls is further compounded by  
6                   the long latency period for cancer?

7                   A. Well, it's made difficult by several  
8                   factors, that is one of them, to identify populations  
9                   that may act as appropriate controls. There are  
10                  several things that may make that more difficult. I  
11                  agree that that is one of them.

12                  Q. And that could include, for example,  
13                  multiple opportunities for exposure to other possible  
14                  substances that might be carcinogenic?

15                  A. You mean is that a problem -- does  
16                  that create difficulties in identifying a control  
17                  population?

18                  Q. Yes.

19                  A. Well, that creates lots of  
20                  difficulties for either -- for both the control and the  
21                  study population. But generally, yes, I would agree  
22                  with that.

23                  Q. Thank you. Would it also be fair to  
24                  say that the effects of other exposures on rates of  
25                  cancer are rarely known and sometimes can have more

1 than an additive effect?

2 A. You are talking now generally still?

3 Q. Yes.

4 A. And I guess your question is, if you  
5 are studing -- I guess I don't understand your  
6 question. There seemed to be two.

7 Q. If you are focusing on one substance  
8 but you also have the possibility of multiple exposures  
9 to others, does that affect your understanding of the  
10 rate of cancer and, in particular, does it affect  
11 the -- whether in fact the effect you are observing can  
12 be more than additive?

13 A. I don't think so. In a typical study  
14 of an occupational group that's exposed to a chemical,  
15 if you are interested in that one chemical, it is  
16 almost always the case that those same workers are  
17 exposed to other chemicals because lots -- most of,  
18 say, manufacturing involves multiple chemical  
19 exposures.

20 You try to match those against some  
21 control group that is identical in all respects except  
22 they don't have exposure to the chemical you are  
23 interested in or they have a very much reduced  
24 exposure. That's a very hard thing to accomplish and  
25 that's the reason these studies are not controlled.

1                   Now, you do that the best you can and you  
2 may observe an excess of, say, cancer in that worker  
3 group. Now, what you can say about the excess then is  
4 limited by the fact that they had multiple chemical  
5 exposures. So you could say, well there seems to be an  
6 excess of cancer in this occupation, but we are not  
7 sure of what it is due to. That would be very hard to  
8 figure out.

9                   Now, whether the excess you see involves  
10 some additive or multiple effect of mixtures of  
11 chemicals, you can't tell from that kind of study.  
12 That requires much more sophisticated investigation.  
13 There are such cases, but you can't tell it from a  
14 simple single epidemiologic study.

15                  Q. Well, I am not particularly  
16 interested in whether we are trying to establish it  
17 from one study only, let's keep it plural.

18                  A. All right.

19                  Q. You raised an issue I just wanted to  
20 follow up on. If an effect is more than additive it is  
21 synergistic; is that correct?

22                  A. That's the general term that is used,  
23 yes.

24                  Q. And perhaps just for the record,  
25 could you just indicate to the Board what synergism is?

1                   A. Agent A causes a risk "x" at a given  
2 level, agent B by itself causes risk "y". An additive  
3 relationship, if you put agent A and agent B together  
4 so both exposures occur, the total risk if they are  
5 additive is "x" plus "y".

6                   There are some cases where when you put A  
7 and B together the risk is more than "x" plus "y".  
8 When it is more it is called a synergistic effect. If  
9 it's just "x" plus "y", that's an additive effect.  
10 There is also antagonism which is less than "x" plus  
11 "y".

12                  Q. So with respect to -- I'm sorry?

13                  A. Okay.

14                  Q. So with respect to synergism, then,  
15 the effect of two agents could be greater than -- when  
16 put together could be greater than the effect of either  
17 of them operating separately?

18                  A. Yes. There is one very good example  
19 of that in cancer and that is the combined effect of  
20 asbestos exposure and smoking.

21                  Q. Okay. Dr. Rodricks, I wonder if we  
22 could just summarize what I think is the gist of what  
23 you have told us so far arising from the questions that  
24 I have asked with respect to cancer.

25                  The factors that can contribute to the

1       insensitivity of human or epidemiologic studies can  
2       include, as I understand your testimony --

3                     A. You said the word insensitivity?

4                     Q. Yes.

5                     A. Okay.

6                     Q. Can include the long latency period  
7       between exposure to an agent and the onset of cancer  
8       and, as we discussed, it could be between 5 and 40  
9       years?

10                  A. Yes.

11                  Q. The high background rate for cancer?

12                  A. Generally -- we didn't cover that,  
13       but if you are trying to deal with cancers that have a  
14       very high prevalence in the population, that reduces  
15       the sensitivity, yes.

16                  Q. And, thirdly, exposure to several  
17       carcinogens over one's lifetime?

18                  A. I'm not sure that reduces  
19       sensitivity, that complicates the problem of finding a  
20       causal relationship to a specific agent. I'm not quite  
21       sure that has to do with sensitivity.

22                  Q. All right, that's fine. Now, I want  
23       to turn to your discussion of the Kansas study, and I  
24       wonder if I could refer you initially to page 53.

25                  I believe we are looking at the top of

1       the page and you indicate there that:

2                 "An odds ratio..." the acronym is OR,  
3                 "...of 1.0 signifies no difference  
4                 between cases and controls for the  
5                 studied exposure."

6                 A. Yes.

7                 Q. And in the -- sorry, let me just go  
8                 through a couple of these portions of this page before  
9                 I get to my first question.

10                In the next paragraph, paragraph 2 on  
11                page 53, you are discussing your opinion that the range  
12                of variation, which is 1.9 to 19.5, in the odds ratio  
13                estimate of risk of contracting non-Hodgkin's lymphoma  
14                is so great that the estimate itself, because it is  
15                based on very few cases, must be considered unstable.  
16                Is that your testimony still?

17                A. Yes.

18                Q. And just so I understand what you  
19                mean by the word 'unstable', Dr. Rodricks, do you mean  
20                that the range of ORs could change with the addition of  
21                extra cases?

22                A. It means that we are dealing with so  
23                few, such a small number of cases here that one more or  
24                less could change that greatly. It's very, very  
25                sensitive to a small change. That's all it means.

1                   Q. Would you agree with me, Dr.

2                   Rodricks, that the chance of an additional case  
3                   dropping the range to 1.0 is equal to the chance that  
4                   one extra case could raise the OR above 19.5?

5                   A. I'm sorry, you are assuming one more  
6                   case is found in this cohort?

7                   Q. Let's assume that.

8                   A. And then the question was, if you did  
9                   find one more case the odds ratio would go greatly  
10                  above six?

11                  Q. Sorry, no, I meant the range. Let me  
12                  restate the sentence -- restate the question.

13                  A. Okay.

14                  Q. Would you agree with me that the  
15                  chance of an additional case dropping the range to 1.0  
16                  is equal to the chance that one extra case could raise  
17                  the range above 19.5?

18                  A. A chance of one more case dropping  
19                  the range to one?

20                  Q. Yes.

21                  A. Assuming that the controls have no  
22                  more cases. The bottom end of the range is not going  
23                  to go -- if that happens it's not going toward one, it  
24                  is going to go the other way.

25                  Q. It's going to get higher?

1                   A. Yes. In other words, there is a  
2 greater likelihood of -- it is a stronger association  
3 if you add one more case, and I can't calculate that  
4 here.

5                   Q. Would you agree with me that it is  
6 highly unlikely that with additional cases the range is  
7 going to centre around 1.0?

8                   A. Well, this observation was -- for  
9 these cases the odds ratio was already six. So if your  
10 assumption is we are going to find more cases and the  
11 control rate is not going to change, the odds ratio is  
12 going to increase and increase and confidence interval  
13 around that is going to shift accordingly and probably  
14 become narrower in fact because you've got more cases.  
15 It is not going to go down to zero.

16                  Q. The data from the Kansas study, Dr.  
17 Rodricks, says that there is only a 5 per cent chance  
18 that the OR is less than 1.0; isn't that right?

19                  A. For this particular finding it says  
20 that there is only a 5 per cent chance that it is less  
21 than 1.9.

22                  Q. All right. There is a 95 per cent  
23 confidence that the OR is between 1.9 and 19.5; is that  
24 right?

25                  A. That's correct.

1                   Q. And you indicate that the range is  
2 above an OR of 1.0; is that right?

3                   A. Yes, that's another way of saying  
4 this is a statistically significant odds ratio.

5                   If the -- you will see in a lot of the  
6 epidemiology studies that the odds ratio is reported  
7 within the confidence limits and if the lower  
8 confidence limit is above one, that generally means we  
9 are talking about a statistically significant  
10 association.

11                  Q. Let's continue with page 2 -- sorry,  
12 page 53 and now look at paragraph -- or Item 2 at the  
13 bottom of that page, and actually the paragraph goes on  
14 to page 54. Let me just read the paragraph into the  
15 record so we understand the context.

16                  "The chi-square test was employed to  
17 evaluate whether or not a trend for  
18 an increasing frequency of herbicide use  
19 existed. A significant trend was found.  
20 This trend is accounted for primarily by  
21 the significant excess risk associated  
22 with the highest frequency of use  
23 category when this experience is compared  
24 with that of nonfarmers. There was no  
25 difference in the ORs for the first three

1                   categories of days of use per year, but  
2                   the two highest categories showed some  
3                   elevations. This suggests that while a  
4                   trend may exist, this may be open to  
5                   question. It is questionable whether the  
6                   establishment of a trend based primarily  
7                   upon an excess in one data point, is  
8                   appropriate and reliable..."

9                   Dr. Rodricks, just so I understand the  
10                  chi-square test, if the Kansas study passed this test,  
11                  would you agree with me that by definition a  
12                  significant trend was established for increasing  
13                  frequency of herbicide use?

14                  A. Well, we are talking about a trend  
15                  here, we are talking about an increase in the rate,  
16                  number of cases, if you like, of non-Hodgkin's lymphoma  
17                  as a function of frequency of use of herbicides, and  
18                  there certainly was an excess in the high exposure  
19                  group, the one defined a little bit earlier with the  
20                  odds ratio of six.

21                  You see, a trend refers to a relationship  
22                  between exposure and the odds ratio. That is, as we  
23                  talked about earlier, likely to be the phenomenon of  
24                  increasing odds ratio with increasing exposure. The  
25                  trend refers to that sort of dose response

1 relationship, not to a single point.

2                   And this is not a specific criticism  
3 here, but simply to point out that finding -- that the  
4 single elevation where others were not elevated is not  
5 strong evidence of a trend in the data. You can't say  
6 it isn't, but it's just not strong evidence.

7                   So trend is the key word here, not -- I  
8 am not questioning that the high exposure category did  
9 have this significant increase odds ratio. That's not  
10 the intent here.

11                  Q. I think I am not clear on what the  
12 intent of your bullet Item 2 then was?

13                  A. Well, we do a statistical test to  
14 determine whether or not you find what is called a  
15 trend in the data; that is, whether you see over a  
16 range of exposures an increasing risk.

17                  Now, the authors conclude that there was  
18 a trend. I simply pointed out -- actually it was one  
19 of my statisticians who pointed this out, it was really  
20 sort of flat and then went up one data point to a high  
21 odds ratio and that didn't seem to be strong evidence  
22 of a trend.

23                  Again, I don't make very much of this.  
24 This is not a terribly important point. There was no  
25 difference in the odds ratios for the first three

1 categories of exposures and then all a sudden you had  
2 an increased odds ratio. That doesn't look like a  
3 trend, even though statistically it satisfies certain  
4 criteria for a trend.

5 Q. The trend is as reliable as the  
6 statistics say it is; is that right?

7 A. I guess I don't understand the  
8 question. You mean is a statistical test for trend the  
9 only determinant of whether this is one?

10 Q. Let me go withdraw that question and  
11 ask you a different one that might clear this up.  
12 Would you agree with me that there was less than a five  
13 per cent chance that the trend seen was only due to  
14 random events?

15 A. By the statistical test, that is  
16 correct.

17 Q. I wonder if you could also confirm  
18 for me that in fact there was less than a .4/100ths  
19 chance that the trend seen was only due to random  
20 events?

21 A. May I look at the exhibit?

22 Q. Exhibit 754.

23 MR. CASSIDY: Do you have that, Dr.  
24 Rodricks.

25 DR. RODRICKS: I do now.

1                   MR. CASSIDY: Do you also have Exhibit  
2                   789?

3                   MR. CASTRILLI: We are looking at page  
4                   1142, table -- sorry, I believe it's Table 1. In any  
5                   event, it's the only table on page 1142.

6                   Q. Dr. Rodricks, I presume you are  
7                   looking at the final column on the right-hand side of  
8                   that table, the non-Hodgkin's lymphoma column?

9                   DR. RODRICKS: A. Yes, I'm looking at  
10                  table -- the only table, as you said, I can't read it,  
11                  on page 1142. I can't read the number of the table.  
12                  And in the table under Non-Hodgkin's Lymphoma they have  
13                  categorized exposures among lymphomas according to the  
14                  frequency that they reported using herbicides in  
15                  general.

16                  Q. Dr. Rodricks, when I look -- I'm  
17                  sorry, were you finished?

18                  A. Yes, I can answer the question. They  
19                  have one, two, three, four five exposure categories if  
20                  you look at that listed in days per year, 0, 1 to 5, 6  
21                  to 10, 11 to 20 and greater than 21. If you look in  
22                  the far right-hand column you will see the odds ratios  
23                  they reported.

24                  Now, for the first four --

25                  Q. Dr. Rodricks, perhaps for the record

1 you can simply identify the four odds ratios you are  
2 referring to so it is clear in the transcript?

3 A. Yes. I am looking under the last  
4 column, Non-Hodgkin's Lymphoma, and for the frequency  
5 of use category zero, the odds ratio was 1.3. Do you  
6 see that?

7 Q. Yes. Please continue.

8 A. Those are farmers. You will notice  
9 that the farmers themselves without any herbicide  
10 exposure had an increase above the non-farmers.

11 Then 1 to 5 days a year, the odds ratio  
12 was 1.4 and if you look at the confidence interval, not  
13 different from the first; 6 to 10 days per year it was  
14 -12.6, again, not different from the first two; 11 to 20  
15 days per year, 2 .6, again statistically not different  
16 from the first three; then the one we were referring to  
17 in the text, more than 21 days per year, it jumped to  
18 six and that one was statistically different from the  
19 1.3 and that is the association in this study that has  
20 caused the concern.

21 Now, when you do a statistical test, a  
22 so-called chi-square test for trend, it is  
23 statistically significant at the .0004 level. That is  
24 highly significant.

25 My statement in the excerpt -- my

1 statement here simply notes that even though that is  
2 statistically significant as a trend and I'm not --  
3 that's quite clear that it is, just note that the first  
4 four exposures were basically flat and then you have a  
5 jump in the odds ratio.

6 That doesn't look like a trend one, would  
7 expect to see some increase in risk with increasing  
8 exposure, but I'm certainly not denying that that is  
9 statistically speaking a trend and certainly strongly  
10 influenced by the odds ratio in the high exposure  
11 group, not by the others.

12 Q. Dr. Rodricks, I am interested in your  
13 use of the term flat. If we look at the five numbers  
14 -you just identified into the record, 1.3, 1.4, 1.6, 2.6  
15 and 6.0, if we were to plot that on a graph would that  
16 look like a flat line to you?

17 A. Well, just remember that the 1.3 and  
18 the 2.6 are indistinguishable statistically by these  
19 tests. Notice the lower confidence limit on both is  
20 .8. Remember, the zero exposure had 94 cases, whereas  
21 when we were dealing with the 11 to 12 days per year,  
22 there are only five cases there. So the confident is  
23 fairly wide.

24 If you were to plot them, they might look  
25 like a gradual increase. They would look like a

1 gradual increase if you plotted them without the  
2 confidence intervals, yes.

3 Q. So it is clearly a trend; isn't that  
4 right?

5 A. I wouldn't call it a trend and I have  
6 seen other real trends in the data, but I'm not going  
7 to argue with you if you prefer to call it a trend.

8 Q. Well, it's your evidence we care  
9 about, Dr. Rodricks, I am not in a position to give  
10 evidence.

11 A. Well, when I've seen instances of a  
12 real trend -- I mean, I've been recently through the  
13 benzene epidemiology data and there you see, in this  
14 case, worker studies, rubble workers exposed to benzene  
15 get leukemia, and there are since several studies one  
16 can see increasing exposure. You get clearly increased  
17 levels of risk.

18 That's a pretty strong trend in the data  
19 or the response relationships. I have seen the same  
20 thing with arsenic exposures by inhalation.

21 Statistically speaking this a trend. I am not going  
22 to -- obviously not going to debate that.

23 Q. Thank you. I think we are still on  
24 page 53 of your evidence. You are discussing on that  
25 page several uncertainties and I just want to clear I

1 understand what it is that you are concerned about  
2 here.

3 When I look at all six of the points you  
4 identify between pages 53 to 57, taken as a whole, is  
5 it your testimony that the Kansas study did not rule  
6 out all the possibley causes of non-Hodgkin's lymphoma?

7 A. Are you on page 54?

8 Q. Actuall, I was taking --

9 A. The conclusions?

10 Q. I was really taking pages 53 through  
11 57 together as a whole. Would a fair statement of your  
12 position as expressed in those pages be that you are  
13 saying the Kansas study did not rule out all other  
14 possible causes of non-Hodgkin's lymphoma?

15 A. In this particular group, that's  
16 correct.

17 Q. And is that what you mean when you  
18 say at page 56 of your evidence -- I guess it really  
19 summarizes it. At the top of the page:

20 "In short, no causal connection between  
21 the use of 2,4-D and increased risk of  
22 NHL was established."

23 A. That's my ultimate conclusion if you  
24 were pointing to 2,4-D specifically, yes.

25 Q. Now, isn't it essentially impossible

1 for any single epidemiology study to meet that  
2 standard?

3 A. Yes, I think I went through that.

4 MR. CASTRILLI: Madam Chair, do we break  
5 at three o'clock?

6 MADAM CHAIR: 3:10, Mr. Castrilli?

7 MR. CASTRILLI: 3:10, I'm sorry. I don't  
8 know why I fix on hours like that.

9 MR. CASSIDY: Just while we are waiting  
10 for Mr. Castrilli, could the witnesses confirm that you  
11 have Exhibit 789?

12 DR. RACHMAN: What is that, Mr. Cassidy?

13 MR. CASSIDY: The letter from...

14 DR. RACHMAN: I am sure I have seen that  
15 here somewhere.

16 MR. CASSIDY: All right, thank you.

17 DR. RACHMAN: Yes. Dated August 28, 1985  
18 to Ms. Kathleen Murphy.

19 MR. CASSIDY: Yes, thank you.

20 MADAM CHAIR: Dr. Rodricks, we've had  
21 testimony from one witness who referred to the fact  
22 that 2,4-D has been used going back to the early 1940s.  
23 It has been the case with some chemical agents that one  
24 problem in studying them is the fact that they haven't  
25 been in use for that long and you have to wait for

1       cancers to develop because they haven't been in the  
2       market or they haven't been used for a long period of  
3       time.

4                   How would you see the very long record of  
5       use of 2,4-D fitting into the possibility that it is a  
6       carcinogen; in other words, the fact that it has been  
7       in use for 40 or 50 years, would you expect to be able  
8       to find cancers more easily because of its widespread  
9       use?

10                  DR. RODRICKS: The general answer to that  
11       is yes, for sure. It came into use I think in about  
12       '47 and was used very widely, so you have 40 plus years  
13       of use.

14                  I guess -- I can't remember or recall  
15       whether in all of these various studies where they  
16       looked at populations what the earliest exposure might  
17       have been in those populations. Some of them go back  
18       into the 50's for sure.

19                  So the latency here, I guess, in some  
20       cases has been quite long. We're not sure in some of  
21       the studies how long people might have been exposed.  
22       That is a bit problematic.

23                  MADAM CHAIR: Nor do we have details on  
24       the formulations, but...

25                  DR. RODRICKS: That's correct. The other

1       thing, I think what is difficult about all this is that  
2       it wasn't just 2,4-D but other phenoxy herbicides that  
3       came into use at the same time, particularly 2,4,5-T,  
4       which is now no longer used and was probably a more  
5       important material during the 50s and 60s and that  
6       confounds interpretation of almost all of these  
7       studies.

8 MR. CASTRILLI: Q. Page 58 of your  
9 evidence. Dr. Rodricks, here you are reviewing the  
10 results and your interpretation of the Western  
11 Washington Study also known, I guess, as the Woods  
12 study in other contexts.

18 DR. RODRICKS: A. Or chlorophenols.

19 They looked at the two combined, chlorophenols are a--

20 Q. Right. For the purposes of --

23 Q. All right, thank you. And as I  
24 understand a further conclusion you draw, which is  
25 expressed at page 59 of your evidence, the Woods study

1 must be considered a negative study with respect to  
2 finding an association between 2,4-D and either NHL or  
3 STS. Is that still your testimony?

4 A. Yes.

5 Q. The Woods study did find increased  
6 risks for those potentially exposed to  
7 phenoxyherbicides in any occupation for 15 years or  
8 more during the period prior to 15 years before cancer  
9 diagnosis; is that correct?

10 A. That was the one increased risk they  
11 found, yes, related to phenoxies in general. I would  
12 just like to add, no information about 2,4-D  
13 specifically at all.

14 Q. I believe that Mr. Cassidy actually  
15 filed that study. You are, of course, familiar with  
16 it; is that right, Dr. Rodricks?

17 A. I have read it, yes. I have studied  
18 it.

19 MR. CASTRILLI: Madam Chair, I would like  
20 to make this the next exhibit. It is entitled Soft  
21 Tissue Sarcoma and Non-Hodgkin's Lymphoma in Relation  
22 to Phenoxyherbicide and Chlorinated Phenol Exposure in  
23 Western Washington, by James S. Woods et al. It  
24 appeared in the journal of the National Cancer  
25 Institute, Volume 78 in May 1987.

1                   MADAM CHAIR: That's Exhibit 1247.

2    ---EXHIBIT NO. 1247: Document entitled Soft Tissue  
3                   Sarcoma and Non-Hodgkin's  
4                   Lymphoma in Relation to  
5                   Phenoxyherbicide and Chlorinated  
6                   Phenol Exposure in Western  
7                   Washington, by James S. Woods et  
8                   al.

9                   MR. CASTRILLI: Madam Chair, I wonder if  
10                  this might be an appropriate place for a break to give  
11                  Dr. Rodricks an opportunity to scan it again. I know  
12                  he is familiar with it, but, in any event, it might  
13                  give him an opportunity to reconsider his position.

14                  Would this be an appropriate place for a  
15                  break?

16                  MADAM CHAIR: Yes, I think you want to  
17                  break, Mr. Castrilli. We will take one right now.

18                  MR. CASTRILLI: All right, thank you.

19                  MR. CASSIDY: Madam Chair, I wonder if I  
20                  could ask if Ms. Devaul could provide me with a copy of  
21                  Exhibit 1233.

22                  Mr. Castrilli has indicated he intends to  
23                  cross-examine the witnesses on this and we have been  
24                  spent the last hour and a half trying to locate our  
25                  copy and we are having some difficulty and if we could  
                      at least borrow an extra one of the Board's copy, the  
                      witnesses will --

26                  MADAM CHAIR: And that's 12...?

1 MR. CASSIDY: 33.

2 MR. CASTRILLI: Madam Chair, it is  
3 another telephone directory size document known as the  
4 Weeks report. Mr. Martel has it in his hand.

5 MR. CASSIDY: I have a feeling what  
6 happened is our exhibit is with the BEAK witnesses who  
7 were cross-examined at length.

8 MR. CASTRILLI: Does that mean it's in  
9 Mexico?

10 MADAM CHAIR: Why don't you take this  
11 copy.

12 MR. CASSIDY: Thank you very much, Madam  
13 Chair.

14 MR. CASTRILLIS: Thank you.

15 MADAM CHAIR: The Board will be back in  
16 20 minutes.

17 ---Recess taken at 3:10 p.m.

18 ---On resuming at 3:35 p.m.

19 MADAM CHAIR: Please be seated.

20 MR. CASTRILLI: Madam Chair, if you will  
21 give me one moment, I think my microphone melted over  
22 the break. I want get it back up to the right  
23 position.

24 Q. Dr. Rodricks, we were discussing  
25 before the break -- or about to begin discussing before

1       the break the West Washington study.

2                     DR. RODRICKS: A. Yes.

3                     Q. And your commentary on it in your  
4       evidence, which is at pages -- or is found at pages 58  
5       and 59. And I have now introduced as an exhibit the  
6       study that was under discussion, the Woods study and  
7       that is now Exhibit 12...

8                     MADAM CHAIR: 47.

9                     MR. CASTRILLI: 1247.

10                  Q. Just as a preliminary matter, can you  
11       confirm for me that the -- I will call it the Woods  
12       study just so you will understand what I mean by that.

13                  I understand that the Woods study  
14       specifically found that estimated risks of NHL were  
15       elevated among forestry herbicide applicators. Is that  
16       your understanding?

17                  DR. RODRICKS: A. Well, that's one of  
18       many, many findings in the study, not the only one.

19                  Q. I wasn't suggesting it was the only  
20       one, but it is you're understanding that that is one of  
21       the findings of the study; is that correct?

22                  A. Yes.

23                  Q. And can you confirm --

24                  A. You did say forestry--

25                  Q. Yes, I did --

1                   A. --because there are many other  
2 spraying occupations with no elevated risk. Did you  
3 say forestry or farmland?

4                   Q. I said forestry.

5                   A. Okay. Yes, with forestry they did  
6 find high elevation.

7                   MR. MARTEL: Can we repeat it, please?

8                   MR. CASTRILLI: I'm sorry, Mr. Martel,  
9 were you asking me to repeat the question so we are  
10 sure about Dr. Rodricks' answer?

11                  MR. MARTEL: Yes.

12                  MR. CASTRILLI: Okay, I will do that.

13                  Q. Dr. Rodricks, can you confirm for me  
14 .that the Woods study specifically found that estimated  
15 risks of NHL, that's non-Hodgkin's lymphoma, were  
16 elevated among forestry herbicide applicators?

17                  DR. RODRICKS: A. That is correct.

18                  Q. Thank you. And would you agree with  
19 me, Dr. Rodricks, that spraying forests with  
20 phenoxyherbicides gave the highest risk? Is that your  
21 understanding?

22                  A. Well, among the occupational risks  
23 that they looked at, spraying forests with herbicide in  
24 general gave the highest risk, yes.

25                  Q. And we see that --

1                   A. That's just among the occupational  
2                   risks.

3                   Q. Yes, and that's the context in which  
4                   the question was asked. And we see that reflected, for  
5                   example, at page 899 in the abstract, in the middle of  
6                   the abstract. And more specifically, would you agree  
7                   with me, Dr. Rodricks, we see it reflected at page --  
8                   Table 4 at page 903?

9                   A. Yes, Table 4 is where they summarize  
10                  their analysis of risks associated with various  
11                  occupations in which phenoxyherbicides are used and  
12                  they categorize the occupation or activity in that  
13                  table according to whether they believe it is  
14                  relatively low, medium or more intense exposure to the  
15                  herbicides, then they list the odds ratio, as you can  
16                  see, for soft tissue sarcoma and NHL.

17                  Q. All right. And if we look at the  
18                  heading for NHL under phenoxyherbicides and we go down  
19                  the column to spraying forests with herbicide, this is  
20                  under the high exposure occupation, the finding was an  
21                  odds ratio of 4.80? That would be 4.8 times the risk  
22                  of someone not exposed; is that your understanding of--

23                  A. That's correct.

24                  Q. --how to read Table 4?

25                  A. That's correct.

1 Q. Thank you. Can I refer you in  
2 Exhibit 1247, that's the Woods study -- I'm sorry, we  
3 are still with the Woods study, to page 907.

4 MADAM CHAIR: One question, Dr. Rodricks.  
5 What was the total study population?

6 DR. RODRICKS: This was called a case  
7 control study where they selected cases of people with  
8 non-Hodgkin's lymphoma, who had it or had died from it.  
9 The total number of cases is listed here.

10 MADAM CHAIR: So is that the 128 with STS  
11 and the 576 with --

12 DR. RODRICKS: Yes, they had 576 NHL  
13 cases.

14 MADAM CHAIR: And the per cent study  
15 population column refers to the number of forestry  
16 workers being one per cent of--

17 DR. RODRICKS: Yes, the size of the  
18 various worker--

19 MADAM CHAIR: --the 576?

20 DR. RODRICKS: --populations that you see  
21 in Table 4, you could multiply that per cent by the  
22 total number of cases to get the number of NHL cases  
23 considered there.

24 MADAM CHAIR: So it's one per cent?

25 DR. RODRICKS: That's why the

1       confidence -- if you look at the confidence intervals  
2       on the 4.8 figure they are fairly wide. I guess that's  
3       probably six cases.

4                     MADAM CHAIR: Okay, thank you.

5                     MR. CASTRILLI: Q. Dr. Rodricks, we are  
6       now turning our attention to page 907 of the Woods  
7       study.

8                     DR. RODRICKS: A. Yes.

9                     Q. Under this is under the general  
10      section of the report dealing -- well, it's the  
11      authors' discussion of their findings or a portion of  
12      their discussion with respect to their findings.

13                  And would you agree with me, Dr.  
14      Rodricks, that the Woods study indicates that it may be  
15      hard to find a control population because dioxins and  
16      furans are so widespread in American and Canadian  
17      populations and that this may account for the  
18      differences in risk estimates between the Woods study  
19      and the Swedish studies?

20                  A. I didn't get to this part of the  
21      discussion. I don't recall it very well. I would  
22      really need to read it again.

23                  Q. Why don't we read it together.

24      Looking at column two on page 907?

25                  A. Yes.

1                   Q. The paragraph that begins:

2        "Differences..."

3                   A. Yes.

4                   Q. Do you see it?

5                   A. Yes.

6                   Q. Maybe I will just read it into the  
7 record.

8                   "Differences in risk estimates observed  
9                   between this and the Swedish studies  
10                  might also be accounted for on the basis  
11                  of variation in the extent of  
12                  non-occupational exposure received by the  
13                  general populations in the areas where  
14                  the studies were conducted. Several  
15                  investigators..." and these are referred  
16                  to in references 50 and 51,

17                  "...have recently reported widespread  
18                  contamination of the general population  
19                  in the United States and Canada with  
20                  PCDDs...."

21                  Just stopping there, Dr. Rodricks, that  
22                  would be dioxins?

23                  A. Yes, that's the broad category of  
24                  polychlorinated dioxins. Yes.

25                  Q. "...and PCDFs..." That would be the

1 broad category of furans?

2 A. That's correct.

3 Q. "...based on analysis of human fat  
4 samples. The findings indicate that  
5 although higher levels of total dioxins  
6 and other contaminants may be seen in  
7 some exposed persons, there is  
8 considerable overlap in actual tissue  
9 concentrations of such substances between  
10 some person's with confirmed occupational  
11 exposures and others who are not  
12 previously known to have been exposed  
13 through job related activities. These  
14 observations suggest that epidemiologic  
15 studies conducted in areas where the  
16 extensive use of phenoxyherbicides and  
17 chlorophenols has occurred may have  
18 inadvertently included subjects who have  
19 experienced significant exposure to the  
20 chemicals of concern outside of the  
21 occupational setting. Should this be the  
22 case, it is possible that estimates of  
23 actual risk based on recall of  
24 occupational exposures alone may be  
25 underestimated, owing to non-differential

misclassification of subjects according  
to exposure status."

3                           And there is a reference 52 at the end of  
4                           that paragraph.

5 A. Yes.

6                           Q. In general, Dr. Rodricks, do you  
7 agree with the statement I have just read into the  
8 record?

9                   A. Well, I don't -- I thought I knew  
10        pretty well the data on background levels, so-called;  
11        that is, levels of these materials and people not  
12        occupationally exposed. There are in the five to ten  
13        parts per trillion range in most of us.

14 The statement I am wondering is  
15 'considerable overlap'. Let me see the exact words.

16 Q. Sorry, perhaps you could direct  
17 our --

18                   A. "...considerable overlap in actual  
19                   tissue concentrations of such subjects  
20                   between some persons with confirmed  
21                   occupational exposure and others not  
22                   occupationally exposed."

I would have to see the data they are referring to there. That doesn't strike true to me. Occupational exposures tend to be considerably higher,

1       but let me say, what they are trying to do here is to  
2       explain the difference in observation, the striking  
3       difference in observation here from Sweden.

4                   I see most of this discussion as setting  
5       forth some hypotheses about why such a difference might  
6       exist. They are assuming the dioxins or furans might  
7       be involved in this without any real evidence that in  
8       fact they are. It is a perfectly reasonable  
9       hypothesis, but it is nothing more than that.

10                  If it's true, we would have to agree with  
11       its conclusion that you could obscure a risk if your  
12       background population indeed had in the U.S. and Canada  
13       a higher background risk. That would be true. But  
14       this is very conjectural and it is more hypothesis  
15       generated.

16                  Q. With that caveat, Dr. Rodricks, do  
17       you agree with the paragraph?

18                  A. If it is true that dioxins are  
19       causally related to these conditions and if it is true  
20       that the general level and background population does  
21       overlap with the occupational background level, and I  
22       am skeptical about that, but if that is true, that  
23       would tend to reduce the sensitivities of these tests  
24       in the U.S. or Canada to protect effects. That would  
25       be true, but only if the first conditions are correct

1 and I am not sure I accept that.

2 Q. Would you agree with me, Dr.

3 Rodricks, that the assessment that I just read into the  
4 record suggests possible problems with the Woods  
5 studies overall conclusion that you rely upon?

6 In particular, let me continue so you  
7 understand the context of the question. You state  
8 that the Woods study did not find an increase in the  
9 risk of either STS or NHL among users of  
10 phenoxyherbicides.

11 Would you agree with me that the  
12 assessment that I just read into the record at page 907  
13 suggests possible problems with the Woods study overall  
14 conclusion that you rely upon, in particular the one I  
15 just suggested?

16 A. I think I would go so far as to say  
17 that if this were correct, which you read in the  
18 paragraph, it might explain why an excess might be  
19 found in Sweden and not here. That does not  
20 necessarily create -- that is not a problem in the  
21 Wood's study, it is an attempt to explain the  
22 difference in findings.

23 You are saying that the Woods study would  
24 have less sensitivity to detect effect than would the  
25 Swedish studies. It is not the fault of the study.

1                   Q. Isn't the possibility raised in that  
2 paragraph, Dr. Rodricks -- I apologize, I almost called  
3 you Dr. Woods.

4                   Isn't the possibility raised in this  
5 paragraph, Dr. Rodricks, that the Woods studies  
6 estimates of risk of cancer from phenoxyherbicides  
7 including 2,4-D may have been underestimated?

8                   A. If this is all true, I agree, yes,  
9 relative to what was estimated in Sweden.

10                  Q. And that's because of the confounding  
11 factors described in the passage I just read to you; is  
12 that right?

13                  A. That's correct.

14                  Q. Let's continue with page 907. We are  
15 now looking at the last paragraph on the page. I am  
16 not going to read the entire paragraph, it goes on for  
17 some time on to page 907. I am just going read the  
18 first portion of that paragraph.

19                  "To estimate the..."

20                  Sorry, do you have the paragraph? It is  
21 the one that begins: "To estimate..."

22                  A. Yes, I see it.

23                  Q. "To estimate the extent to which  
24 non-occupational exposure to  
25 phenoxyherbicides may have occurred in

1                   the present investigation, we have  
2                   evaluated data from several air  
3                   monitoring studies..." and the authors  
4                   refer to references 53 and 54 in this regard,  
5                   "....conducted during the spraying season  
6                   in the Pacific Northwest. These data  
7                   indicate that phenoxyacetic acids..."  
8                   Just stopping there there. Dr. Rodricks,  
9                   my understanding is that's another terminology for  
10                  phenoxyherbicides?

11                 A. Yes.

12                 Q. "...as well as PCDDs...

13                 That's the general category of dioxins?

14                 A. Yes.

15                 Q. "...can transported in the atmosphere  
16                 either as vapour or adsorbed on particles  
17                 for distances ranging from several  
18                 hundred feet up to a mile from the  
19                 application area..."

20                 There is a further reference, reference  
21                 54,

22                 "...depending on weather conditions and  
23                 mode of dispersion."

24                 Now, just stopping there, Dr. Rodricks,  
25                 is that an assessment you agree with?

1                   A. The reference is a study I have not  
2 read, reference 54.

3                   Q. Do you have any better information?

4                   A. No.

5                   Q. And let's just hypothesize again, if  
6 we might for a moment, Dr. Rodricks. Would you agree  
7 with me that if this is true, then this would again  
8 underscore the difficulty in finding a control  
9 population because these chemicals are so widespread in  
10 the general non-occupational human population?

11                  A. If the assumption is that the dioxins  
12 are what are responsible here for any observed  
13 excesses, then this would be correct. What you say  
14 would be correct.

15                  MADAM CHAIR: Excuse me, Dr. Rodricks.  
16 Why wouldn't the assumption work equally well in the  
17 other direction, that there would be -- that a control  
18 would in fact control for everyone, for all the  
19 populations exposure to any dioxins?

20                  DR. RODRICKS: Well, I guess if I think  
21 it's true, the assumption here is that if individuals  
22 have collected in their body dioxins, which remain in  
23 the body, but the phenoxyacetic acids do not, they are  
24 rather quickly excreted, but if dioxins are accumulated  
25 in the body there would be an exposure there.

1                   It is not zero in that population, but if  
2       it increased and there's potentially higher background  
3       risk, if you like, in the so-called controls, if the  
4       risk is really due to these dioxins such that -- that  
5       would tend to obscure a difference between controls,  
6       because they are not zero controls, they have some  
7       finite body burden as against people who have clearer  
8       and obvious occupational exposures.

9                   MR. CASTRILLI: Q. Dr. Rodricks,  
10      continuing with you. Would you agree with me that risk  
11      estimates like the ones in the Woods study, based only  
12      on assessment of occupational exposures without asking  
13      about residential or home use exposures, could be  
14      underestimated as a result of exposure in this  
15      classification.

16                  DR. RODRICKS: A. Home use exposures,  
17      yes, that could be a confounding factor in the control  
18      rooms.

19                  Q. If I could just refer you to page 908  
20      in this regard. We are looking at a slightly -- sorry,  
21      we are looking at what would be the left-hand column on  
22      the page, slightly more than halfway down the sentence  
23      beginning: "Nevertheless... "

24                  Do you see that?

25                  A. No. 908?

1                   Q. Sorry, page 908, left-hand column,  
2 top left-hand portion of the page, halfway down that  
3 paragraph begins the phrase:

4                   "Nevertheless, should phenoxyherbicides  
5 and/or their contaminants..."

6                   Do you see that?

7                   A. I'm sorry.

8                   Q. Let me just point it out to you.

9                   A. Yes, okay.

10                  Q. The Woods study authors indicate:

11                  "Nevertheless, should phenoxyherbicides  
12 and/or their contaminants increase the  
13 risk of cancer at environmental exposure  
14 levels or, as recently suggested, produce  
15 subclinical immune system alterations  
16 that may predispose to such risks, it is  
17 possible that risk estimates based solely  
18 on assessment of occupational exposures  
19 could be attenuated as a result of  
20 exposure misclassification."

21                  Do you agree with that assessment?

22                  A. If all the premises are true, I don't  
23 agree that all the premises are true. Their  
24 hypothesizing what would result if, for example, there  
25 were an accumulation of contaminants, such as dioxins,

1       in people who are not occupationally exposed and it  
2       could alter immune systems, if that is true, and I  
3       don't believe there is convincing evidence to support  
4       that, but if that were true then there could be an  
5       attenuation of risk -- of the risk estimates, assuming  
6       that only the occupational exposures contribute to  
7       disease. So the conclusions would be true if all the  
8       premises are correct, but I don't agree with all the  
9       premises.

10           I also have to note here, and it is  
11       something I didn't remember on my first reading on your  
12       last question, that they did test this hypothesis about  
13       home use of phenoxyherbicides. I just noticed this, if  
14       I may just go back to the previous--

15           Q. Please do.

16           A. --two sentences. They eliminated  
17       subjects or they redid an analysis apparently who  
18       reported home use exposures to phenoxyherbicides and  
19       chlorophenols in the present study and that had no  
20       effect on the estimated risks for either STS or NHL to  
21       paraphrase that sentence. Do you see the sentence?

22           Q. Yes.

23           A. Hence, it is unlikely that bias due  
24       to such exposures could account for the large  
25       differences in risk estimates observed between these

1 and the Swedish studies. I had not recalled that they  
2 had tried to do that, but that pertains to your last  
3 question -- the question before last.

4 Q. Now, at page 59 of your evidence.

5 A. I'm sorry, may I -- I'm sorry, please  
6 go ahead.

7 Q. At page 59 of your evidence you are  
8 summarizing your position with respect to the Woods  
9 study and you state, this would be the second paragraph  
10 on the page:

11 "However, because the direct analysis of  
12 phenoxyherbicides did not show an  
13 association between this exposure and  
14 disease, this must be considered a  
15 negative study with respect to finding an  
16 association between 2,4-D and either NHL  
17 or STS."

18 I gather that's still your position; is  
19 that right?

20 A. Yes, and that is based on all of the  
21 data in this paper.

22 Q. Can I refer you in the Woods study to  
23 page 901.

24 A. Yes.

25 Q. We are looking at the second full

1 paragraph.

2 A. "In coding occupational exposure...."

3 Q. Yes. The paragraph begins with that  
4 phrase.

5 A. Yes.

6 Q. Firstly, can you confirm for me, Dr.  
7 Rodricks, that the relative risks in the Woods study  
8 are determined on a theoretical estimation of whether a  
9 certain occupation is associated with low, medium or  
10 high exposure?

11 A. Excuse me one moment. Now, what is  
12 your question? Theoretically...

13 Q. Sorry, let me repeat the question.  
14 Can you confirm for me that the relative risks in the  
15 Woods study are determined on a theoretical estimation  
16 of whether a certain occupation is associated with low,  
17 medium or high exposure?

18 A. Well, they collected information for  
19 each person on the job held and the use of  
20 phenoxyherbicides and chlorophenols. They made an  
21 assumption that there couldn't have been exposure prior  
22 to the date when they were first marketed. They say  
23 that in this paragraph, and then they say:

24 "The coding of each job episode held by a  
25 study subject according to intensity

1                   and duration of exposure..."

2                   That was the coding they did when they  
3                   collected the information,

4                   "...permitted evaluation of the exposure  
5                   history of each subject in terms of  
6                   duration of continuous or cumulative  
7                   exposure at each dose level. Thus a  
8                   complete exposure profile on each subject  
9                   for each class of chemical under  
10                  evaluation was obtained."

11                  That's all they say on that topic. I  
12                  take their word that that's what they did.

13                  Q. I think it might have been helpful if  
14                  I had directed you to another passage in the paper.  
15                  Let me do that now.

16                  A. All right.

17                  Q. Page 900.

18                  A. All right.

19                  Q. We are looking at the right-hand  
20                  column near the bottom. We see the heading -- sorry,  
21                  we see the sentence that begins: "Each job title..."

22                  Do you see that?

23                  A. Yes.

24                  Q. I will just read that into the  
25                  record.

1                   "Each job title or activity was then  
2                   assigned to a 'high', 'medium', 'low' or  
3                   'no' exposure category for both  
4                   phenoxyherbicides and chlorophenols,  
5                   reflecting the consensus of the  
6                   consultant group of the likely intensity  
7                   of exposure to each chemical received in  
8                   that occupation. Examples of  
9                   occupational activities in each  
10                  exposure category are given in the  
11                  tables."

12                 A. That is correct. That is quite  
13                 typical of these kinds of retrospective studies.  
14                 That's quite typical.

15                 Q. Now, let's just look at the paragraph  
16                 I referred you to earlier which is the second full  
17                 paragraph on page 901, the paragraph beginning: "In  
18                 coding occupational exposure..."

19                 Do you have that?

20                 A. Yes.

21                 Q. "In coding occupational exposure  
22                 to phenoxyherbicides..." It says:  
23                 "...based on job descriptions..."

24                 Is that your understanding, the coding  
25                 was done on the basis of job descriptions?

1                   A. Yes, and you see those in Table 4.  
2                   That is the actual job descriptions.

3                   Q. Let's look at Table 3 first, which is  
4                   on page 902 at the bottom of the page.

5                   A. Yes.

6                   Q. If we just look, Dr. Rodricks, at the  
7                   italics associated with that table -- let me just read  
8                   that into the record.

9                   " Risk (pooled odds ratio) of developing  
10                  STS or NHL in men...exposed...20-79..."  
11                  that's 20 years to 79 years,

12                  "...by estimated intensity of past  
13                  occupational exposure to  
14                  phenoxyherbicides..."

15                  Just stopping there, Dr. Rodricks. Can  
16                  you confirm for me that the relative risks in the Woods  
17                  study are determined on a theoretical estimation of  
18                  whether a certain occupation is associated with low,,  
19                  medium or high exposure and not on whether the  
20                  interviewee reported personal low, medium or high  
21                  exposure?

22                  A. Right, the interviewee would have no  
23                  way to know that, no one has any way to know that and  
24                  that's why they turned to consultants who understand  
25                  these jobs and the activities and, again, this is

1 fairly typical in occupational studies where you are  
2 trying to reconstruct historical exposures. There is  
3 nothing much else you can do.

4 Q. So would it be fair to say, Dr.  
5 Rodricks, that what the Woods study did was determine  
6 exposure by job title and not by interview?

7 A. Well, they determined occupational  
8 history by interview job categories and then they  
9 ranked -- and also asked questions about the use of  
10 phenoxyherbicides and other chemicals, chlorophenols,  
11 and then ranked them using consultant experts who know  
12 about these occupational jobs categories and the  
13 relative intensities of exposure.

14 Q. Well, just let me be clear on your  
15 response to my question. What the Woods study did was  
16 determine exposure by job title; is that right?

17 A. Yes, and that's true of almost every  
18 study there is on these herbicides.

19 Q. And would it be fair to say, Dr.  
20 Rodricks, that the Woods study consultants assigned  
21 people to particular exposure categories by occupation  
22 and not by reported or demonstrated exposure?

23 A. Well --

24 Q. I'm sorry, let me finish the question  
25 so you understand the context. In fact, let me repeat

1           the question so you understand the context.

2                         Would you agree with me that Woods  
3                         assigned people to particular exposure categories by  
4                         occupation and not by reported or demonstrated exposure  
5                         and he pooled all of these folks together in terms of  
6                         their exposures?

7                         A. We could just go back to what we have  
8                         already read into the record. They used a  
9                         questionnaire to identify occupations and activities  
10                         that involved the use of phenoxyherbicides and/or  
11                         chlorophenols. They used consultants to identify those  
12                         categories of jobs and they identified, as it says here  
13                         in the paragraph you referred to on page 900, 34  
14                         specific job titles, 17 job activities that involved  
15                         potential exposure to phenoxyherbicides or  
16                         chlorophenols, and then they categorized those  
17                         according to intensity of exposure that would be  
18                         associated with those kinds of jobs or activities.

19                         So, yes, I think we have said this  
20                         already. That's what they did.

21                         Q. I want the record to be clear on  
22                         this. Let me restate it a slightly different way and  
23                         see if your answer still is the same.

24                         Woods pooled exposures together for all  
25                         those with only one time exposure or a few exposures to

1 many exposures and he put those within a category; is  
2 that right?

3 A. Would you repeat that?

4 Q. I'm sorry?

5 A. Say that again.

6 Q. Yes. Woods pooled exposures together  
7 for all those with only one time exposure, a few  
8 exposures to many exposures and put those within a  
9 category; in other words, he might have had -- if you  
10 can graphically see this, he might have put a range of  
11 exposures within the low category, he might have put a  
12 range of exposures within the medium category and he  
13 put a range of exposures in the high category and we  
14 don't know what those are; is that right?

15 A. No, he doesn't say anymore than we  
16 relied upon the work of the consultants to do that.

17 Q. Would you agree with me, Dr.  
18 Rodricks, that serious dilution would occur by lumping  
19 cases together on the basis of their exposure -- excuse  
20 me, on the basis of their occupation rather than on  
21 their reported recalled exposure? Is that a  
22 possibility?

23 A. It's possible if there are serious  
24 areas made in the exposure categorization, yes.

25 Q. Let's continue with page 901. This

1       is the first full paragraph on page 901. As I  
2       understand what the Woods consultants did from a  
3       reading of the study, is that they asked the  
4       interviewees about their extent of exposure to specific  
5       chemicals of interest and the precise time intervals  
6       during which each exposure episode had occurred.

7                   That's your understanding as well; is  
8       that right?

9                   A. That's what they attempted to do,  
10      yes.

11                  Q. Having done that, would you agree  
12      with me, Dr. Rodricks, that nowhere in Woods study are  
13      any relative risks calculated on the basis of these  
14      responses, whatever they were, because Woods does not  
15      in fact report on what they were?

16                  A. Can you give me a few moments here?

17                  Q. Sure, please take your time.

18                  A. My assumption in all of this analysis  
19      was that -- the three paragraphs we referred to  
20      describe the total activity they went through to  
21      categorize workers' exposure based on job categories  
22      and interview in which people described their jobs, job  
23      titles and other information they could collect, and  
24      then they assigned people according to that collective  
25      informing, and that the analyses, insofar as it deals

1       with exposure versus risk, included that total  
2       analysis. That's been my interpretation all along  
3       here.

4                   Q. Do you want another moment to review  
5       the document?

6                   A. Your question is whether the  
7       categories in Table 3, low, medium and high were based  
8       strictly on information supplied by the consultants?

9                   Q. I wouldn't --

10                  A. Is that your question?

11                  Q. I wouldn't put it that way. Let me  
12       rephrase the question I asked earlier and see if we can  
13       shorten this up.

14                  A. Please.

15                  Q. The proposition I was putting to you,  
16       Dr. Rodricks, was that nowhere in the Woods study are  
17       any relative risks calculated on the basis of these  
18       responses, the responses we referred to at page 901,  
19       whatever they were, because Woods does no report on  
20       what these were; isn't that your understanding?

21                  A. My understanding is that that's what  
22       the categories in Table 3 refer to.

23                  Q. The relative risks?

24                  A. The odds ratios, mm-hmm.

25                  Q. Where are the relative risks referred

1 to?

2 A. Well, under NHL, for example, the  
3 exposure category. I am referring to Table 3 on page  
4 902. The odds ratio is another name for relative risk.

5 Q. Sorry, I think we are two ships in  
6 the night here and it probably my fault in terms of the  
7 question. Where in the Woods study do the authors  
8 calculate relative risks on the basis of the responses  
9 they received?

10 A. Well, they do several different kinds  
11 of analyses based on those responses. This is one.

12 Q. From the interviewees. Where do we  
13 have the information from the interviewees in terms of  
14 relative risks?

15 A. In Table 3 they constructed an  
16 exposure category based on information about job title,  
17 length of time in the job and information provided by  
18 the interview and they state they were interviewed  
19 1983-85, and then in Table 4 they have odd ratios for  
20 the same group of men, but they break them out a  
21 different way according to specific occupations or  
22 activities and those occupations or activities are  
23 broken into low, medium and high exposure categories.

24 Q. Dr. Rodricks, what I am going to do  
25 is I am going to red flag that question and come back

1 to it because I think if we move a little further into  
2 the Woods study it will become more apparent and then  
3 we can come back to it.

4 A. We have some miscommunication here, I  
5 don't understand your question.

6 Q. All right. Looking at page 901, this  
7 is the second full paragraph in the middle of the page.

8 A. Yes.

9 Q. We are looking about halfway down in  
10 that paragraph, the sentence that begins: "The coding  
11 of each job episode..."

12 Do you see that? The word 'the' begins  
13 on the right-hand -- sorry, the left-hand column, the  
14 last word in the sentence. Let me assist you, if I  
15 might.

16 A. "The coding of each job episode...."

17 Q. Yes, that's right. Let me just read  
18 that into the record and we will pursue this.

19 "The coding of each job episode held by a  
20 study subject according to intensity and  
21 duration of exposure permitted evaluation  
22 of the exposure history of each subject  
23 in terms of duration of continuous or  
24 cumulative exposure at each dose level."

25 Now, would you agree with me, Dr.

1       Rodricks, that if Woods in that paragraph I just read  
2       into the record is referring to reported exposure this  
3       is never in fact evaluated statistically in the Woods  
4       study?

5                   A. I assume that's what Table 3 is. Is  
6       there some reason why -- point out to me why that would  
7       not be the case.

8                   Q. Because I think from the body of the  
9       study it is clear that he was reporting exposure. As  
10      he says in Table 3, for example, estimated on the basis  
11      of job categorization, not on the basis of actual  
12      exposure testing or assessment -- not testing,  
13      assessment.

14                  A. Well, it says this was based on  
15      interviews and I don't know why you would collect all  
16      that information in interviews and go to all that  
17      trouble and then not use it.

18                  I have been under the assumption the  
19      exposure category, as listed in the Table B, would  
20      represent that full analysis of job title and  
21      information about exposures reported during --  
22      collected the interviews. It says these were cases and  
23      controls interviewed in the '83 to '85 period. So my  
24      assumption is that's what they did.

25                  Q. Let me just --

1                   A. If that's wrong, I guess I don't know  
2 why it would be wrong.

3                   Q. Let me just ask you, then, your  
4 understanding of where Woods is reporting reported --  
5 sorry, your understanding of where Woods statistically  
6 evaluates reported exposure is to be found in which  
7 table?

8                   A. Table 3 is one evaluation, Table 4 is  
9 another evaluation and then there are several other  
10 evaluations of odds ratios in the text for different --  
11 they looked at many different kinds of exposures and  
12 occupations.

13                  Q. Let me put this proposition to you,  
14 Dr. Rodricks. If Woods is talking about theoretical  
15 exposure classification assigned to each occupation,  
16 such a breakdown for each subject does not appear in  
17 the statistic calculations.

18                  I am thinking, for example, if you look  
19 at Table 4 on page 903, we don't have a category such  
20 as spraying woodlands with herbicides high exposure  
21 versus spraying woodlands with herbicides brief or low  
22 exposure. Would you agree with me we don't have that  
23 kind of breakdown in Table 4?

24                  A. Within some of the job categories?

25                  Q. Yes.

1                   A. They were unable to determine that.

2                   Q. Your answer is...?

3                   A. They were unable to determine that,  
4 presumably.

5                   Q. Thank you.

6                   MADAM CHAIR: Excuse me, Mr. Castrilli, I  
7 don't want to interrupt your cross-examination, but on  
8 page 901 they do talk about some work that they did  
9 with recall bias in terms of exploring specifically  
10 with the interviewees where they couldn't be clear  
11 about their exposure profiles and so they specifically  
12 asked them for more information about it, specifically  
13 for recall bias, but it must have helped refine a bit  
14 what the specific kinds of exposures were in the  
15 categories.

16                  DR. RODRICKS: Yes.

17                  MR. CASTRILLI: Madam Chair, what I am  
18 exploring with this witness is whether in fact Woods  
19 every reports on that in the body of the study, or  
20 whether all he reports is the categorizations based on  
21 the job title descriptions the consultants designed.

22                  DR. RODRICKS: Well, my understanding is  
23 that the exposure categories as set forth in Table 3  
24 and otherwise described in the text and in Table 4, a  
25 different way to look at them, was based on the

1 information collected from consultants and people who  
2 were interviewed, combined information.

3 MR. CASTRILLI: Q. Can I refer you to  
4 page 903 of the Woods report.

5 DR. RODRICKS: A. Yes.

6 Q. Can you confirm for me, Dr. Rodricks,  
7 that Woods calculated relative risks associated with  
8 exposure specifically to 2,4-D and 2,4,5,-T and to  
9 phenoxyherbicides in general as he indicates on the  
10 left-hand column?

11 A. He reports somehow breaking out  
12 information on 2,4-D and 2,4,5-T and phenoxies in  
13 general and he calculates risks of .73, that is less  
14 than one for 2,4-D, .98 for 2,4,5-T and .87 for  
15 phenoxyherbicides in general.

16 What is not all clear is how he broke out  
17 2,4-D. You can't tell from this paper how he was able  
18 to separate the two. He does say that there is no real  
19 basis for it given in the paper that I could see.

20 Q. And would you agree with me that he  
21 apparently pooled everything from one time exposure to  
22 heavy exposure for the calculations?

23 A. The overall calculation? The .87  
24 figure for phenoxyherbicide includes the entire study  
25 population, yes.

1                   Q. And what we have in Table 4 on page  
2     903 are estimates or potential exposure and not  
3     necessarily actual exposure; is that right?

4                   A. Yes, we have in none of the studies,  
5     positive or negative, any estimate of actual exposure.

6                   Q. I just want to be clear about one  
7     last point with respect to this study.

8                   The Woods study does not determine  
9     relative risk on the basis of recalled high, medium or  
10    low exposure; is that your understanding?

11                  A. My understanding is that Table 3, as  
12    I read it, includes all of the exposure information  
13    they collected for interviews and for job  
14    categorization.

15                  Q. And Table 3 talks about estimated  
16    exposure?

17                  A. Surely they are estimated.

18                  Q. Do you know whether Woods gathered  
19    data on the frequency of use within a particular  
20    occupation?

21                  A. Well, that he does not report and I  
22    assume he did not do that or he would have reported it.

23                  In the occupation categories; that is,  
24    Table 4, he just had broad categories and groups them  
25    himself. Maybe that is what you were getting at. He

1       does using consultants group the job categories, if  
2       that's what you mean, by high, medium or low exposure,  
3       but there is no breakdown within those specific job  
4       categories, that is correct.

5                   I have assumed, and I don't see any  
6       reason why it is wrong, that Table 3 would reflect  
7       categorization by total intensity of exposure.

8                   Q. Dr. Rodricks, with all of the  
9       questions about the Woods study we have been discussing  
10      for the the last few minutes, I would like to have your  
11      very clear evidence on the record as to why you believe  
12      this is a negative study?

13                  A. Well, I don't believe it shows an  
14       excess of NHL or STS related to exposure to 2,4-D. The  
15       total evidence in the study does not show an excess.  
16       I am not by any means saying this proves that these are  
17       not carcinogens, not by any means. I am just saying it  
18       does not show an excess.

19                  The data in Table 3 where exposures are  
20       categorized for phenoxyherbicides in general, the odds  
21       ratios are listed there for STS and NHL and by their  
22       categorization there is no significant increase in the  
23       three exposure groups over the uncontrols, any of  
24       those.

25                  There are other -- we went over the data

1       on the entire population where the odds ratio was .87.  
2       According to occupation; that is, if you look at Table  
3       4, there is one occupation in which we have -- I'm  
4       sorry, two occupations in which you have a  
5       statistically significant excess: the farmer  
6       categorized as medium exposure where the odds ratio was  
7       1.33, was barely significant, spraying forest with  
8       herbicides was clearly significant although a  
9       relatively small number of cases, but nevertheless  
10      significant, and all the others -- in none of the  
11      others is there any excess at all.

12                  As a matter of fact, the strongest excess  
13      for NHL occurred in Table 6, even these are -- you have  
14      be be very careful here too. A small part of the  
15      population, but the strongest excess you would find in  
16      Table 6 are those individuals who have some compromised  
17      immune system. People who have been taking  
18      immunosuppressant drugs have a relative risk of 10.9.

19                  They looked at these other factors, too,  
20      and I certainly wouldn't conclude that they prove that  
21      immunosuppressant drugs cause NHL either, not by any  
22      means. There are some elevated NHLs associated with  
23      chemicals, chlordane and DDT in combination. That's in  
24      Table 7, welding metal fumes, those kinds of industrial  
25      exposures. Those are a few excesses, but altogether I

1 find this unconvincing with respect to -- certainly  
2 with respect to 2,4-D and even with the  
3 phenoxyherbicides altogether.

4 I just emphasize that. I don't want mean  
5 to prove that they are not carcinogens, that's not my  
6 implication in my statement, but this does not add  
7 anything to the evidence.

8 Q. With all of the confounding factors  
9 associated with this particular study, why isn't it  
10 simply inconclusive as opposed to negative?

11 A. Negative here means no excess shown,  
12 no significant association shown altogether and maybe  
13 that would be a better way to say it. Negative may  
14 be -- if by negative you are reading that this shows it  
15 not to be a carcinogen, then negative is wrong. I  
16 didn't mean it that way.

17 Q. I am wondering, Dr. Rodricks, if we  
18 might refer -- I haven't talked about your evidence for  
19 a while, I'd almost forgotten the principal document we  
20 are here to focus on.

21 I wonder if I could direct your attention  
22 to your exhibit again, it is Exhibit 1239, and we are  
23 looking at the -- beginning at page 60.

24 A. What number is this?

25 Q. I'm sorry, your witness statement is

1                   Exhibit 1239.

2                   A. Oh.

3                   Q. Some of these numbers become  
4                   significant for some of us during the course of the  
5                   hearing and we forget that they mean absolutely nothing  
6                   to witnesses who come and go.

7                   We are looking initially Dr. Rodricks, at  
8                   page 60.

9                   A. Yes.

10                  Q. And we are looking at the last full  
11                  paragraph on that page.

12                  A. Yes.

13                  Q. You begin -- well, I will begin the  
14                  sentence in the middle of -- sorry, let me start by  
15                  comment again.

16                  Let me just read the entire sentence into  
17                  the record so it is clear what we are talking about:

18                  "...if the Swedish studies are not  
19                  included in the plot of probability  
20                  distributions of the ORs from the case  
21                  control studies, then the remaining  
22                  studies are found to cluster close to  
23                  unity, indicating no difference between  
24                  cases and controls in phenoxyherbicide  
25                  use with respect to STS or NHL."

1                   And just stopping there, Dr. Rodricks.

2       You refer in this regard to Figure 1, which is found at  
3       page 612, and then let me just continue with the  
4       remainder of the comment I want your views on.

5                   "Figure 1 illustrates the relative size  
6                   of the ORs from the five NHL studies  
7                   reviewed by Bond..."

8                   And, Madam Chair, Bond is of course  
9       Exhibit 715 in these proceedings.

10                  "The comparison is somewhat crude in  
11                  that the definition of exposure was not  
12                  uniform for these studies. In addition,  
13                  the Hardell study combined NHL cases with  
14                  HD..."

15                  HD is Hodgin's disease; is that right?

16                  A. Yes.

17                  Q. "...so at the least, the confidence  
18                  limits would be wider if the report  
19                  focused upon NHL. Figure 1 demonstrates  
20                  that a causal relationship between  
21                  phenoxyherbicide use and cancer use has  
22                  not been established. Three of these  
23                  studies (Woods et al. 1987..."

24                  That's now exhibit 1247,  
25                  "...Cantor et al. 1986, and Pearce et al.

1                   1986) are negative (ORs cluster about  
2                   1.0). Of the two positive studies, the  
3                   methodology of the Hardell et al. (1981)  
4                   study is open to question, while the  
5                   results of Hoar et al..." also known as  
6                   the Kansas study, "...are not specific for  
7                   phenoxyherbicides."

8                   Now, I would just like to focus on the --  
9                   initially on the first sentence of the last paragraph  
10                  on page 60 of your evidence where you say:

11                  "...if the Swedish studies are not  
12                  included in the plot of probability  
13                  distributions of the ORs from the case  
14                  control studies, then the remaining  
15                  studies are found to cluster close to  
16                  unity, indicating no difference between  
17                  cases and controls in phenoxyherbicide  
18                  use with respect to STS or NHL."

19                  Now, just stopping there. Unity equals  
20                  1.0; is that my understanding?

21                  A. Yes.

22                  Q. Now, let's look at Figure 61 --  
23                  excuse me, Figure 1 on page 61 of your evidence. Now,  
24                  Dr. Rodricks, if we do what you suggest on page 60 and  
25                  we eliminate --

1                   A. Excuse me, this is what Dr. Bond did  
2                   in his paper.

3                   Q. Am I right --

4                   A. I am describing the Bond paper.

5                   Q. Do you adopt his evidence -- or do  
6                   you adopt his methodology?

7                   A. I may have some questions about what  
8                   he did, it's crude in some respects, but it's useful in  
9                   others and I think that's what we describe here.

10                  Q. All right. Let's just focus on  
11                  Figure 1 and if you want make any qualifications that  
12                  you think are appropriate you can -- please do that.

13                  Now, would you agree with me that if we  
14                  were to eliminate the Hardell studies from Figure 1  
15                  that that would still leave four ranges that cluster  
16                  close to 1.0, but even the average -- sorry, that  
17                  cluster closer to 1.0, but even the average of them is  
18                  still greater than 1.0?

19                  A. Well, we must include the confidence  
20                  limits and perhaps a clearer demonstration is in the  
21                  Bond paper itself. Can we refer to that?

22                  Q. We are going to.

23                  A. Okay. The confidence limits for the  
24                  combined four that remain all go in the low end below  
25                  one. You combine them. That's clearer perhaps in the

1 Bond paper itself.

2 Q. Well, would you agree with me that  
3 even if we eliminate the Hardell studies from Figure 1  
4 every study listed on Figure 1 is elevated above 1.0?

5 A. Not statistically. Three are not and  
6 one is.

7 Q. With respect on the odds ratio, Dr.  
8 Rodricks?

9 A. Well, you have to look at the  
10 confidence interval, that's what the bar represents.

11 Q. Well, let's just look at the odds  
12 ratio for a moment. Would you agree with me that with  
13 respect to the odds ratio every other study -- sorry,  
14 and leaving Hardell aside, every study on that page is  
15 elevated above 1.0?

16 A. That's correct.

17 Q. Thank you.

18 Q. Let's look at the Bond paper.

19 MADAM CHAIR: What is the exhibit number  
20 of the bond paper, Mr. Castrilli?

21 MR. CASTRILLI: Actually, it is Exhibit  
22 715 and I am looking for my copy of it.

23 Q. Dr. Rodricks, it might be easier if  
24 we began at page 174 of Exhibit 715.

25 DR. RODRICKS: A. Yes.

1                   Q. This is a table that Bond has put  
2                   together dealing with a number of the studies that you  
3                   have included in Figure 1. In referring to the Kansas  
4                   study, Dr. Rodricks, Bond indicates that the odds ratio  
5                   for the 170 NHL population based cases was 2.2 with a  
6                   95 per cent confidence limit of between 1.2 and 4.1.

7                   Is that how you read Table 1 with respect  
8                   to the Kansas study?

9                   A. Yes, sir, that's for the entire  
10                  population study.

11                  Q. Actually, I'm sorry, what I should  
12                  have done is asked you to get your copy of Exhibit 754  
13                  at the same time. That's the Kansas study. I don't  
14                  know if your copies have numbers on them.

15                  Just looking at the Kansas study, Dr.  
16                  Rodricks, this is Exhibit 754 --

17                  MR. CASSIDY: Do you have that, Dr.  
18                  Rodricks?

19                  DR. RODRICKS: Yes, I have it.

20                  MR. CASTRILLI: Q. Could you help me  
21                  find where the Kansas authors report an odds ratio of  
22                  2.2.

23                  MR. MARTEL: What table are we looking  
24                  at, Mr. Castrilli?

25                  MR. CASTRILLI: Mr. Martel, we are

1 looking at Exhibit 754, the Kansas study, and I have  
2 not referred the witness to a particular page. I would  
3 like him to help me.

4 DR. RODRICKS: I can refer you in Exhibit  
5 754 to page 1143, the table there. Bond derived that  
6 figure from the table. I can't read the number of the  
7 table.

8 MADAM CHAIR: Table 2.

9 DR. RODRICKS: Is it Table 2?

10 "Every used phenoxyacetic acids..."  
11 So it is the overall figure for the  
12 study. 2.2 odds ratio with a confidence interval of  
13 1.2 to 4.1, that's what they use.

14 MR. CASTRILLI: Q. Now, doesn't Bond  
15 assume for the purposes of their analysis that the  
16 results of the five studies represent the same  
17 underlying population at risk?

18 DR. RODRICKS: A. Well, they make the  
19 assumption, but they also qualify that in their text  
20 and they recognize that there is a certain crudeness in  
21 this analysis. They probably do not result the same  
22 underlying distribution.

23 Q. But isn't that assumption important  
24 for Bond in order to permit him to average the case  
25 control studies?

1                   A. Yes, they average the studies by  
2 giving them equal weigh and that assumes they come from  
3 the same -- as the statisticians call it, underlying  
4 distribution of diseases, and we don't know whether  
5 that's true, that is correct. They state that as an  
6 assumption in their analysis.

7                   Q. There is no basis for that  
8 assumption, is there?

9                   A. Well, if in fact -- I don't know  
10 whether there is or not. If in fact these excessess  
11 all represent the very same biological phenomenon, then  
12 there could be, but we don't know whether that's true.

13                  Q. The studies that Bond refers to, at  
14 least in theory, looked at populations exposed to  
15 different levels of risk, isn't that right, in terms of  
16 duration and frequency?

17                  A. Different levels of...

18                  If you mean there are diverse populations  
19 in terms of their exposure patterns and risk patterns,  
20 yes, they are.

21                  What they were attempting to do was do  
22 something a little more than just kind of a qualitative  
23 weight of evidence study, we have used that term  
24 before. In looking at all of the data combined, they  
25 tried to do something a little bit more qualitative

1 here with the odds ratios in the various studies.

2 It is crude, as we say, on page 62 of our  
3 report, but it was an attempt to do something here,  
4 somewhat more systematic than simply looking at all the  
5 data and making a qualitative judgment. I hope in our  
6 report we qualified our confidence in this analysis.

7 Q. Yes, I think you indicated twice it  
8 was crude and once it was misleading.

9 A. Well, it could be. The important  
10 thing about it, I think, is what it seems to show,  
11 crude as it is, is that you have basically one study  
12 with this extraordinarily high risk report in Sweden  
13 which seems to not match the total body of evidence  
14 very well.

15 You have not seen that same striking  
16 phenomenon in other studies. I mean, that's -- maybe  
17 what they have done is kind of a roundabout way of  
18 getting to that conclusion which might be obvious just  
19 upon inspection, but they tried to do it a little bit  
20 more systematically than others have.

21 Q. We've had a discussion this afternoon  
22 about the Woods study which in your evidence you  
23 describe as negative and we've talked about the  
24 possible confounding factors in the Woods study, so  
25 isn't there some difficulty in Bond doing what he did

1       in relation to the Wood study in suggesting there is no  
2       basis for a relationship between what was found in  
3       Sweden and what might have been found in western  
4       Washington?

5                   A. Well, keep in mind that one basis for  
6       the difference between Sweden and Western Washington is  
7       that 2,4-D or the phenoxyherbicides or the dioxins do  
8       not cause cancer. That's one possible explanation for  
9       the observation.

10                  The authors, Woods and others were  
11       looking for other possible explanations for the  
12       difference and we don't know why the difference exists.  
13       All of these studies that Bond looks at have  
14       methodological limitations. I hope we made clear by  
15       now that every epidemiology study does, and they're  
16       different from one study to the other.

17                  Q. Now, in your evidence you discuss the  
18       Canadian farm operator mortality study. Actually, I  
19       guess it is not called that anymore. What is the  
20       title?

21                  MR. CASSIDY: The title is rather  
22       lengthy, Madam Chair.

23                  MADAM CHAIR: Did we make this an exhibit  
24       yet, Mr. Cassidy?

25                  MR. CASSIDY: Yes, it is Exhibit 1244.

1                   MADAM CHAIR: 1244. Thank you.

2                   MR. CASSIDY: Do you intend to break at  
3 five o'clock tonight, Madam Chair?

4                   MADAM CHAIR: Yes, Mr. Cassidy.

5                   MR. CASSIDY: Thank you.

6                   MR. CASTRILLI: Q. Dr. Rodricks, we  
7 have -- sorry, in looking at the Wigle study, being  
8 Exhibit 1244, would you agree with me that in this --  
9 this is a cohort study, as I understand it, of male  
10 Saskatchewan farmers, significant dose response  
11 relationships were noted between risk of non-Hodgkin's  
12 lymphoma and acres sprayed with herbicides.

13                  DR. RODRICKS: A. Yes. You are reading  
14 from the abstract?

15                  Q. Yes. Well, actually it is found in  
16 several places in the report, that's pme place it's  
17 found.

18                  A. Yes.

19                  Q. And the the significant dose response  
20 relationship was with respect to dollars spent on fuel  
21 and oil for farm purposes for 1970.

22                  Would you agree with me, Dr. Rodricks,  
23 that during the period of exposure in this study the  
24 authors indicate that 2,4-D constituted over 90 per  
25 cent and 75 per cent by weight of all herbicide active

1 ingredients used agriculturally in Saskatchewan?

2 A. I would need to look that up, sir.

3 Q. Page 580, left-hand column. I guess  
4 it is the second full paragraph on the page, left-hand  
5 column.

6 A. I'm sorry, on page...

7 Q. 580.

8 A. Oh, sorry. I am afraid my copy is  
9 missing 580. No wonder I am confused.

10 Q. That would make it difficult to find  
11 it.

12 A. As a matter of it is missing every  
13 other page. This was the copy I was given during  
14 lunch.

15 MR. CASSIDY: This one has got them call.

16 MR. CASTRILLI: By your counsel, I would  
17 note.

18 MR. CASSIDY: --my photocopying was not  
19 that bad.

20 MR. CASTRILLI: Did I hear the sound of  
21 an exhibit being torn up?

22 MR. CASSIDY: The witness' copy. The  
23 official exhibit I believe is with Mr. Martel's  
24 safekeeping.

25 DR. RODRICKS: I found the paragraph,

1 yes.

2 MR. CASTRILLI: Q. You don't have any  
3 better information with respect to that study; do you?

4 DR. RODRICKS: A. Certainly not.

5 Q. And the herbicide 2,4,5-T -- sorry I  
6 am just reading from the next sentence in that  
7 paragraph.

8 "The herbicide 2,4,5-T was apparently  
9 used infrequently in agriculture in  
10 Saskatchewan at this time, although it  
11 was in regular use for brush control on  
12 non-crop land."

13 There are several references. Now, you  
14 don't have any better information than that, I presume;  
15 is that right?

16 A. No, I do not.

17 Q. Now, Dr. Rodricks, do you have  
18 Exhibit 717 which was the abstract that was produced in  
19 August of 1989 by Dr. Ritter with respect to this study  
20 which, of course, at that time hadn't been written?

21 A. I'm afraid I don't have that  
22 abstract. It is not identical to the one in the  
23 report?

24 Q. That's what we are going to explore.

25 A. I do not have the abstract.

1                   MADAM CHAIR: Mr. Castrilli, why are we  
2                   going to explore whether this abstract is the same as  
3                   the abstract in the published article?

4                   MR. CASTRILLI: Well, for one reason,  
5                   because I don't believe they are the same and for a  
6                   second reason, Exhibit 717 has been on the record for  
7                   roughly 12 -- 10 months. I believe there was  
8                   commentary on the record with respect to the abstract  
9                   last year and I just want to get the record clarified.

10                  Madam Chair, actually noticing that it is  
11                  almost five o' clock, what I am suggest to suggest is  
12                  that the witnesses be given a copy of the abstract  
13                  from -- sorry, that would be Exhibit 717.

14                  Q. One other item. Dr. Rodricks, you  
15                  will notice if you look at the front page of Exhibit  
16                  1224--

17                  DR. RODRICKS: A. Yes.

18                  Q. --under the heading of Editorials,  
19                  You will see in the second listed item there is an  
20                  editorial by Aaron Blair, Herbicides and Non-Hodgkin's  
21                  Lymphoma, New Evidence From a study of Saskatchewan  
22                  Farmers. Do you have a copy of that?

23                  A. I don't.

24                  MR. CASTRILLI: Madam Chair, I would like  
25                  to make a copy of the item I have just referred to from

1       exhibit 1244 available to the witness for him to  
2       consider overnight and I would suggest that that's  
3       where we resume tomorrow morning.

4                     MADAM CHAIR: All right. One thing, Mr.  
5       Castrilli. Why don't you tell Dr. Rodricks what the  
6       difference is that you see. It shouldn't take long to  
7       clear this up.

8                     MR. CASTRILLI: No, it won't. Dr.  
9       Rodricks, perhaps overnight you could simply review the  
10      first paragraph of Exhibit 717, which is the abstract,  
11      and just advise the Board whether you find that  
12      paragraph is still a conclusion of the authors or  
13      whether it is no longer a conclusion of the authors as  
14      reported in Exhibit 1244.

15                  DR. RODRICKS: Whether the first  
16      paragraph of the abstract...

17                  MR. CASTRILLI: In Exhibit 717 is still a  
18      part of --

19                  DR. RODRICKS: The conclusions of this  
20      paper.

21                  MR. CASTRILLI: The conclusions from  
22      Exhibit 1244. It is an exercise that should not take  
23      very long. I will now make a copy available of the  
24      editorial from Aaron Blair and I would suggest that  
25      that is where we leave matters for this afternoon.

1                   MR. CASSIDY: Do you have a copy you are  
2                   going to give to him right now?

3                   MR. CASTRILLI: Yes, I am going to make a  
4                   copy available to him.

5                   MR. CASSIDY: All right.

6                   MADAM CHAIR: Are we going to make that  
7                   an exhibit, Mr. Castrilli?

8                   MR. CASTRILLI: Yes.

9                   MADAM CHAIR: Exhibit 1248.

10                  ---EXHIBIT NO. 1248: Editorial entitled Herbicides and  
11                   Non-Hodgkin's Lymphoma, New  
12                   Evidence From a study of  
                     Saskatchewan Farmers by Aaron  
                     Blair.

13                  MR. CASSIDY: It may be necessary for me  
14                  to speak to the witnesses, Madam chair, to assist them  
15                  in finding a copy of Exhibit 177, and that's all I  
16                  intend to do.

17                  MADAM CHAIR: Here is my copy. You can  
18                  have a copy run off...

19                  MR. CASSIDY: I also can advise, Madam  
20                  Chair, on this matter that arose over the course of  
21                  lunch, that prior to the completion of the  
22                  examination-in-chief the witnesses were asked to be  
23                  provided with a transcript that refers to another  
24                  exhibit that Mr. Castrilli -- relates to another  
25                  exhibit that Mr. Castrilli advises he intends to

1       cross-examine on, Exhibit 789.

2                     The transcript volume is 125 and I've now  
3       located it and I intend to provide that to the  
4       witnesses as well.

5                     MADAM CHAIR: Any objections?

6                     MR. CASTRILLI: No, not at all.

7                     DR. RACHMAN: Before we break, could I  
8       please ask for a clarification of which materials  
9       exactly you want us to look at this evening to make  
10      sure that we have a complete list.

11                  MR. CASTRILLI: Yes. Madam Chair, I've  
12      made a number of articles available to the witnesses  
13      that are not yet exhibits in which we will be  
14      discussing tomorrow morning. I intend to examine them  
15      on those documents.

16                  With respect to Exhibit 1233, which I  
17      identified earlier today as an exhibit I might wish to  
18      raise with the witnesses, I can advise at this time  
19      that I don't think it will necessary for the panel to  
20      actually look at Exhibit 1233.

21                  DR. RACHMAN: What was Exhibit 1233?

22                  MR. CASTRILLI: Sorry, 1233 is the  
23      phonebook in front of you.

24                  DR. RACHMAN: Oh, the one I have already  
25      read?

1                   MR. CASTRILLI: If you've read it between  
2 midday and now...

3                   MADAM CHAIR: So how many different  
4 pieces of documentation do they have to read tonight,  
5 Mr. Castrilli?

6                   MR. CASTRILLI: Well, the remaining  
7 documentation has not yet been filed with the Board.  
8 I've given them roughly a half a dozen pieces of paper.

9                   MADAM CHAIR: Now, can you direct them  
10 more carefully in their reading so they can answer  
11 succinctly--

12                  MR. CASTRILLI: I think the --

13                  MADAM CHAIR: --tomorrow what the  
14 questions are going to be.

15                  MR. CASTRILLI: Well, I think they should  
16 read the entirety of the documents. They are not very  
17 long in most cases. Some of them are one page long.

18                  DR. RACHMAN: What about 1237?

19                  MR. CASTRILLI: I think we've dealt with  
20 1238 already on the record today and I don't believe  
21 there is anything further that needs to be referred to  
22 with respect to that exhibit.

23                  And just to be fair to the witnesses,  
24 with respect to one document I've provided to them,  
25 which is called the Record of Decision, Pacific

1 Northwest, they need only look at page 6.

2 MR. CASSIDY: That's Exhibit 1236, I  
3 think.

4 MR. CASSIDY: No, that's another one I  
5 have just given them--

6 MR. CASSIDY: Excuse me.

7 MR. CASTRILLI: --that is not yet an  
8 exhibit.

9 DR. RACHMAN: Page 6 only?

10 MR. CASTRILLI: Page 6 of the Record of  
11 Decision, Pacific Northwest.

12 Everything else is comparatively short,  
13 Madam Chair. There is one item that's a little bit  
14 longer, but it is pretty straightforward and they won't  
15 have much difficulty with it.

16 I would like at this time to make the  
17 Aaron Blain editorial the next exhibit.

18 MADAM CHAIR: Thank you. We said that  
19 would be Exhibit 1248.

20 MR. CASTRILLI: Madam Chair, we will be  
21 resuming at 8:30 tomorrow morning?

22 MADAM CHAIR: Yes. When will you be  
23 finished your cross-exam, Mr. Castrilli?

24 MR. CASTRILLI: Madam Chair, I anticipate  
25 being completed by the midday break, lunch time.

1                   MADAM CHAIR: Lunch time.

2                   MR. CASTRILLI: Thereabouts.

3                   MADAM CHAIR: All right. And Ms. Kleer  
4                   is to follow Mr. Castrilli, so she should be here by...

5                   MR. CASTRILLI: I would say late morning  
6                   at the earliest. Thank you, witnesses.

7                   MADAM CHAIR: Mr. Freidin?

8                   MR. FREIDIN: Madam Chair, I am just  
9                   wondering if I could just address the Board for a  
10                  moment, just take a couple of moments.

11                  I assume that the Board has heard about  
12                  the announcements by the government last week that Dr.  
13                  Peter Pearce of the University of British Columbia has  
14                  been retained by the government to perform certain work  
15                  which, I understand, will lead to certain  
16                  recommendations to the Minister of Natural Resources  
17                  concerning an appropriate process for the development  
18                  of an overall forest policy for Ontario.

19                  MADAM CHAIR: I haven't heard anything  
20                  about this, Mr. Freidin.

21                  MR. FREIDIN: All right. On that basis,  
22                  that's why I rose, I felt that as a courtesy to the  
23                  Board I should bring this to the Board's attention and  
24                  I would like to provide you with certain documentation  
25                  which was released along with that announcement, not to

1 suggest that you don't have enough reading to do as it  
2 is.

3 I don't believe these need be marked  
4 exhibits, Madam Chair, but the documents I would like  
5 to give you and Mr. Martel are as follows: The  
6 curriculum vitae of Dr. Pearce, the terms of reference  
7 for Dr. Pearce dated May the 10th, 1990, and a letter  
8 from the Minister. It is standard form letter from the  
9 Minister of Natural Resources which was sent out to a  
10 large number of groups and individuals, and I can  
11 assure you that that large group included parties at  
12 this particular proceeding.

13 MADAM CHAIR: So give it to us in a  
14 nutshell, Mr. Freidin, what does all of this mean?

15 MR. CASTRILLI: Madam Chair, I wonder if  
16 Mr. Freidin could advise when this was released to the  
17 public?

18 MR. FREIDIN: I believe on the day it  
19 indicated on the terms of reference. I think May the  
20 10th. I can't be precise about -- I would have to  
21 check on the precise date.

22 The reason I provided this to the Board,  
23 Madam Chair, is that it is a matter of -- it may be a  
24 matter of interest to the Board as it is dealing with a  
25 related or complementary matter; that is, an overall

1 policy for forestry in Ontario, it is not limited to  
2 timber management at all.

3 The reason I wanted to bring it to your  
4 attention is that I felt if you heard about it sort of  
5 via grapevine you might ask the question as to what  
6 relationship, if any, it has to what the Board is  
7 doing, whether it affects your ability to do all the  
8 things that we have indicated in past submissions the  
9 Board can do. I didn't want that question to sort of  
10 come to your mind if you heard about it through the  
11 grapevine and wonder what the answer to that was.

12 So I just wanted to provide you with the  
13 material that I have and perhaps just refer you to the  
14 letter from the Minister which indicates that it is  
15 certainly the Ministry's view, on page 2, if you look  
16 at the fourth paragraph and the first sentence, that's  
17 all I need refer to, the Minister has stated that she  
18 wants to emphasize to the people that -- she is  
19 advising about this development. She wants to  
20 emphasize that this policy and review will not  
21 duplicate or pre-empt the ongoing environmental  
22 assessment dealing with timber management.

23 So, again, I just felt as a matter of  
24 courtesy I should bring this to your attention and  
25 answer a question which I thought might come to your

1        mind if you had heard about it, and I believe perhaps a  
2        review of the material that I provided may be of  
3        assistance.

4                    MADAM CHAIR: Thank you, Mr. Freidin.

5                    MR. HUFF: Mr. Freidin, could I have a  
6        copy of the press release that went out last Wednesday  
7        afternoon?

8                    MR. FREIDIN: I don't have a copy of the  
9        press release.

10                  MR. HUFF: Can I have it tomorrow if you  
11        can get it?

12                  MR. FREIDIN: Sure.

13                  MADAM CHAIR: All right. We will adjourn  
14        until tomorrow morning at 8:30. Thank you.

15

16        ---Whereupon the hearing adjourned at 5:10 p.m.,  
17        to be reconvened Thursday, June 14, 1990 commencing  
18        at 8:30 a.m.

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